DIAGNOSIS AND TREATMENT

OF HEAD INJURIES

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WITH 94 ILLUSTRATIONS



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PREFACE

Head injuries have become of increasing importance to physicians and surgeons in every community In a large measure this is due to the appalling increase in the number of highway accidents The automobile which is involved in most road accidents has rightly been called a "modern juggernaut"

Practitioners everywhere are constantly called upon to render emergency treatment and after-care to the victims of automobile accidents falls and other mishaps which result in head injuries

There has been so much controversy concerning the proper treatment of these injuries that the average medical attendant is frequently uncertain as to the best procedure to follow in any case

This book attempts to present a practical and concise approach to the problem Should it prove to be of assistance to the physician or surgeon of average training our efforts will have been amply rewarded

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S. W. G.
W. E

NEW YORK, N Y

INTRODUCTION

Cranial surgery began probably with the treatment of wounds At any rate the Edwin Smith papyrus (earliest known surgical treatise dating from 3000-2500 B C), describes various types of injuries to the head and their treatment. They have continued to furnish an important subject of medical discussion throughout recorded history We can suppose that they were of interest in prehistoric times as well, since men must have discovered very early that an effective way to put an enemy out of action was to strike him a heavy blow on the head. But archeologists have disclosed also that in prehistoric times operations were performed on the cranium for other reasons than the treatment of wounds From survivals of this practice in remote regions such as the Atlas Mountains of Morocco it is believed that these prehistoric trephinings were made as a treatment of epilepsy doubtless on the theory that the subject was possessed and that the demon must be given an opening through which to escape. However if such operations were ever performed in Europe knowledge of them was lost and cranial surgery continued to be practically confined to the treatment of wounds until relatively recent times

Since very little was known about the functions of the intracranial organs early discussions were concerned mainly with the various types of fracture of the skull and the mechanism of their production Until very recently any treatise on injuries of the head would be filled with illustrations of fracture-lines and discussions of why the fractures ran in just those directions But with increasing knowledge of the func

tions of the brain, a change has gradually taken place so that today a similar treatise, such as the present one, is concerned mainly with the intracranial contents and very little with the fractures of the skull for reasons which the authors make abundantly clear.

The increase in knowledge of the functions of the brain which was initiated by the investigations of Broca, and of Fritsch and Hitzig, resulted ultimately in the localization of a brain tumor by inference from the symptoms alone The brilliant feat of Bennett, who induced Godlee to operate and prove the correctness of his conclusions, started a general interest not only in cerebral function but also in cranial surgery and most of all in brain tumors Since that date cranial surgery has tended more and more to concern itself with tumors of the brain, except for the short period of the World War. This interest reached its peak in the dominating personality of Harvey Cushing

But meanwhile an increasingly important factor was renewing interest in wounds of the head. As the automobile furnished faster and faster transportation which exceeded the ability of the human reaction-time to avoid disaster, more and more injuries of the head occurred and county medical societies demanded ever more discussions of their management. In an attempt to diminish the slaughter, laws have been passed to limit the speed at which automobiles may be driven in certain regions but, while manufacturers are permitted to make cars with speeds up to 100 miles an hour, it has proved futile to attempt to impose on drivers by law a speed-limit of 35 miles It seems probable, then, that the subject of injuries to the head will long be one of frequent interest to every physician

By the time that the automobile began a typical American mass-production of head-injuries, the increase of our knowl-

edge of intracranial physiology made it obvious that the injury to the intracranial contents was almost always much more important than that to the skull For this reason we miss in modern treatises the long discussions of the mechanism of fracture This shift in interest is reflected also in the manner of handling these cases No longer are seriously injured patients kept waiting for x ray photographs of the skull to be made these are made only for definite reasons after all symptoms of shock have disappeared, often just before discharge simply because they may be needed as evidence in court. The fracture being usually in good apposition will heal without treatment its position is important mainly to indicate the possible source of continued bleeding

On the other hand the handling of cerebral injuries has been much perfected particularly methods of controlling the increase in intracranial tension By means of intravenous hypertonic solutions, lumbar punctures and eventually decompressive operations the otherwise fatal hypertension can usually be controlled It seems rather generally agreed that operations except for the evacuation of localized collections of blood, had better be avoided. If they are performed one should not be satisfied with halfhearted measures, since the contused brain may increase 50 per cent in volume The usual subtemporal decompression is futile and only increases the damage

The symptoms of patients with injuries to the head change rapidly They should be observed carefully and continuously for several days No patient who has been unconscious even for only a moment should be allowed to go out of reach of a trained observer for the first few days Experience has shown also that it is wiser to put all such patients to bed for two or three weeks their observation is facilitated and disagreeable sequels are thereby avoided.

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sible. For the latter reason the patient s head should be elevated until the pressure of his spinal fluid has returned to normal and from that moment onward his head should be on a level with his body or even lower in some cases until he no longer complains of headache or dizziness He should be allowed up only very gradually, and any complaint of headache and dizziness should return him at once to the horizontal position from which he should not be allowed up to eat, to go to the toilet or for any other reason This treatment is excellent also for the rare malingerer for obvious reasons The rigid enforcement of this management will result in a great reduction in the incidence of the so-called post traumatic neuroses

But when all is said and done, there is much that remains to be learned about head injuries and physicians still have different opinions about their management and about the significance of many symptoms Their honest differences are a source of much unfair criticism by some lawyers who complain that they cannot get the medical men to agree on anything Outside the courtroom physicians more often agree than disagree and when unable to reconcile their views are apt to call an older or more experienced consultant Much of the criticism of physicians would disappear if they made a practice of always collecting their fees before testifying They should probably be made state officials as the judges are they are as expert witnesses, asked to pass judgment

But many matters will remain under discussion for a long time not only in the courtroom but more so at the bedside and in the operating room Clear and concise summaries of the status of medical opinion concerning head injuries such as the following will be needed from time to time especially if the present political unrest widens into a second World War

PERCIVAL BAILEY

CHICAGO, ILLINOIS

DIAGNOSIS AND TREATMENT
OF HEAD INJURIES

“No injury to the head is so slight as to be despised, nor so great as to be despaired of”

—Charles B Nancrede
International Encyclopedia of Surgery

Chapter I

APPLIED ANATOMY OF THE HEAD

THE SCALP

The hair the scalp and the skull provide a protective covering for the underlying vital soft tissue the brain, its membranes and blood vessels Injuries to the head are practically always associated with some degree of laceration contusion or hematoma of the scalp The scalp wound may be of trifling importance but occasionally assumes major significance as for example when an extensive avulsion of the scalp with loss of tissue has occurred making a satisfactory closure difficult. The skin covering the head is peculiarly constructed for the growth of hair and for protection of the vault of the cranium It is thicker than the integument over any other part of the body (0 6 cm over the temporal region to 1 0 cm or more over the occiput) and is closely connected by means of the subcutaneous tissues with the aponeurosis of the occipitofrontalis muscles This aponeurosis commonly called the "galea" is of great importance in cranial surgery

The temporal muscle and the cutaneous muscles of the scalp are made use of in planning the incisions and scalp flaps for cranial operations The occipitofrontalis muscle consists of two fleshy portions the occipital and the frontal, joined by a broad aponeurosis which is continuous with the aponeurosis of the opposite muscle across the vertex. Between the galea and the pericranium there is a layer of loose connective tissue upon which the mobility of the scalp depends This loose areolar tissue enables scalp flaps to be stripped

easily from the pericranium Collections of blood or pus spread freely in all directions in this loose tissue

The temporal muscle (Fig 1) is a fan-shaped structure filling the temporal fossa It is attached to the upper temporal ridge by a white shining fascia, which, together with the muscle, forms an excellent protective covering for the underlying soft tissues when the bone has been removed for purposes of exploration or decompression The scalp is very vascular, most of the blood supply being carried by vessels in the subcutaneous tissue These vessels are terminal branches of the temporal, supra-orbital, posterior auricular and occipital arteries, and pass towards the vertex of the head, running in a tortuous course from their points of origin.

The temporal artery is a continuation of the external carotid It tunnels through the upper part of the parotid gland, and becomes superficial about 5 mm in front of the ear Its importance in surgery of the head lies in the fact that the viability of a scalp flap depends upon its blood supply, and that a flap containing the uninjured temporal artery will have an adequate vascular supply The supra-orbital artery ascends with the supra-orbital nerve from the supra-orbital foramen The frontal artery accompanies the supratrochlear nerve upward between the supra-orbital nerve and the root of the nose The posterior auricular artery ascends with the posterior auricular nerve in a groove behind the mastoid process The occipital artery, along with the great occipital nerve reaches the scalp at a point midway between the external occipital protuberance and the mastoid process It is, in part, because of this great vascularity that incised wounds of the scalp bleed so profusely, as well as the toughness of the scalp which prevents retraction of the cut ends when the arteries are divided The veins of the scalp

are quite large and as a rule bear the same names as the arteries which they accompany

The lymph channels of the scalp are in relation to the principal veins The frontal lymphatics descend from the forehead and eyebrows some going to the submaxillary lymph nodes, while the greater number converge towards the front of the ear where they enter the lymph nodes of the parotid region The parietal lymphatics pass to the mastoid lymph nodes which lie above the insertion of the sternomastoid muscle The occipital lymphatics go partly to the suboccipital nodes in front of the occipital attachment of the trapezius muscle, and partly to the deep cervical lymph nodes under the sternomastoid muscle in the middle of the neck.

The nerves of the scalp (Fig 1) are derived from the trigeminal nerve and the cervical plexus The supra-orbital nerve is a branch of the ophthalmic division of the trigeminal It leaves the orbit by way of the supra-orbital foramen and supplies the skin of the forehead and scalp as far back as the vertex The medial part of the forehead is supplied by the supratrochlear nerve The temporal area of the scalp is supplied by the auriculotemporal nerve, a branch of the third division of the trigeminal The zygomaticotemporal branch of the second division of the fifth nerve supplies a small area between the frontal and temporal regions of the scalp The scalp posterior to the auricle is supplied by branches of the cervical plexus—the great auricular lesser occipital greater occipital and third occipital nerves

THE SKULL

The skull cap or calvarium varies greatly in thickness depending upon the amount of the diploë between the outer and inner tables of compact bone. The inner table of the

skull is thinner and more brittle than the outer, and may sometimes fracture with the outer table remaining intact The average thickness of the skull is 5 mm. The thinnest parts are in the temporal regions, the thickest parts are at the base The bones of the cranium are the frontal, two parietal, occipital, two temporal, the sphenoid, and the ethmoid, and are connected at their edges by sutures The frontal bone develops from two portions which become joined by the frontal suture shortly after birth This suture closes rapidly, so that as a rule it is no longer demonstrable after the age of six years, though rarely it may persist for many years The sagittal suture unites the two parietal bones The parietal bones are connected with the frontal bone by the coronal suture, and with the occipital bone by the lambdoid suture The temporal bones are joined to the parietal bones by the squamous sutures The frontal and temporal bones are of special importance in injuries to the skull Fractures of the frontal bone may extend into the frontal sinus, which may serve as a portal of entry for infection into the intracranial cavity The petrous portion of the temporal bone is often involved in fractures of the base while the middle meningeal artery may be torn in fractures of the squamous portion of the temporal bone

THE DURA MATER, DURAL SINUSES AND SUBARACHNOID SPACE

The dura mater is a tough membrane, and serves as a second line of defense in the protection of the underlying brain when the skull has been injured The dura mater consists of two layers of tough fibrous tissue which in most places are firmly adherent to each other These layers separate in certain places to form venous channels, the sinuses of the dura mater. The dura mater also forms strong partitions, the falx

cerebri and tentorium cerebelli which divide the cranial cavity into compartments, and form supporting structures for the brain The dura mater is firmly attached along the convexity of the cranium by fibrous processes of the outer layer to the midline of the skull. The attachments to the coronal and lambdoid sutures are less firm Along the base, the dural attachments are especially firm at the foramina, where processes of the dura mater blend and become continuous with the sheaths of blood vessels and nerves which pass out of and into the cranial cavity In early life and in old age the dura mater is more adherent to the inner table of the skull

The superior longitudinal sinus begins at the foramen cecum, through which it receives a vein from the nasal cavity and occupies the convex margin of the falx It passes backward to the internal occipital protuberance, where it deviates, usually to the right, and terminates in the corresponding lateral (transverse) sinus The superior longitudinal sinus is triangular in cross section narrow in front, but increasing in caliber as it passes backward. It receives a number of large vessels, the superior cerebral veins, from the brain These tend to arrange themselves in trunks—a frontal, a precentral, a postcentral and occipital. They are relatively unsupported, and may be torn even with minor head injuries The inferior longitudinal sinus occupies the concave margin of the falx cerebri and receives the veins from the mesial surface of the cerebral hemispheres and from the falx cerebri The inferior longitudinal sinus is continued as the straight sinus at the junction of the falx cerebri and tentorium cerebelli The straight sinus continues backward and downward to empty into the lateral sinus of the side opposite the one receiving the main channel of the superior longi

tudinal sinus The lateral sinuses are the main channels into which all the other sinuses converge They begin at the internal occipital protuberance and pass laterally downward and inward in the grooves of the occipital and temporal bones to reach the jugular foramina The diploic veins, mastoid emissary veins, the superior and inferior petrosal sinuses empty into the lateral sinuses The cavernous sinuses are venous plexuses surrounding the body of the sphenoid bone. Through each cavernous sinus the corresponding carotid artery passes Arteriovenous aneurysm may follow a fracture of the base of the skull, which, extending into the sphenoidal fissure, tears the carotid artery. The superior longitudinal and transverse sinuses may be torn in depressed fractures of the vault over these structures The resulting hemorrhage is seldom severe and usually is easily controlled, since the venous pressure in the sinuses is low, especially when the head is elevated

The dura mater is closely applied to the underlying arachnoid membrane and, ordinarily, the subdural space is only a potential space. However, following trauma, the dura mater may be separated from the arachnoid by a collection of blood or fluid, thus creating an actual (subdural) space.

The pia-arachnoid membrane, or leptomeninx, forms a resilient fluid-containing cushion for the brain. The pia mater is in immediate relation to the surface of the brain, containing within its meshes the branching of the cortical vessels. The pia mater follows closely the convolutions of the brain. The outer surface of the leptomeninx is adjacent to the dura mater. Delicate septa pass between the two layers of the leptomeninx The space between (subarachnoid space) is filled by the cerebrospinal fluid.

At the base, where the contour of the brain differs greatly

from that of the dura mater the subarachnoid space becomes wider filling in the discrepancy, and forming the sub arachnoid cisterns The most important cistern is the cisterna magna an enlargement of the subarachnoid space which extends over the dorsal aspect of the medulla oblongata and which is directly continuous with the subarachnoid space of the spinal canal Fluid is removed from this cavity for diag nostic and therapeutic purposes by the suboccipital or cis ternal puncture of Ayer The other important cisterns or reservoirs of cerebrospinal fluid are the cisterna pontis on the under surface of the pons the cisterna interpeduncularis, bounded by the cerebral peduncles the cephalic border of the pons the corpora mamillaria and the tuber cinereum and the cisterna chiasmatis ventral to the optic chiasm and the cisterna ambiens surrounding the midbrain

THE BRAIN

The brain consists of cerebrum diencephalon mesen cephalon pons medulla oblongata and cerebellum (Fig 2) The cerebrum overlies the other portions of the brain, filling the whole of the upper part of the cranial cavity The under surfaces of the frontal lobes rest on the anterior fossae the temporal lobes occupy the middle fossae the under sur faces of the occipital lobes rest on the tentorium cerebelli below which is the cerebellum filling most of the posterior fossa The cerebrum is formed by two symmetrical masses the cerebral hemispheres separated by the falx cerebri and joined by a large transverse commissure called the corpus callosum, and two small transverse commissures the an terior commissure and the transverse fibers of the fornix.

The cerebral cortex is divided by means of sulci and fis sures into frontal, parietal temporal, occipital lobes and the

insula, or island of Reil, which lies at the bottom of the lateral fissure The lateral, or Sylvian fissure separates the temporal lobe from the frontal and parietal lobes, and lies

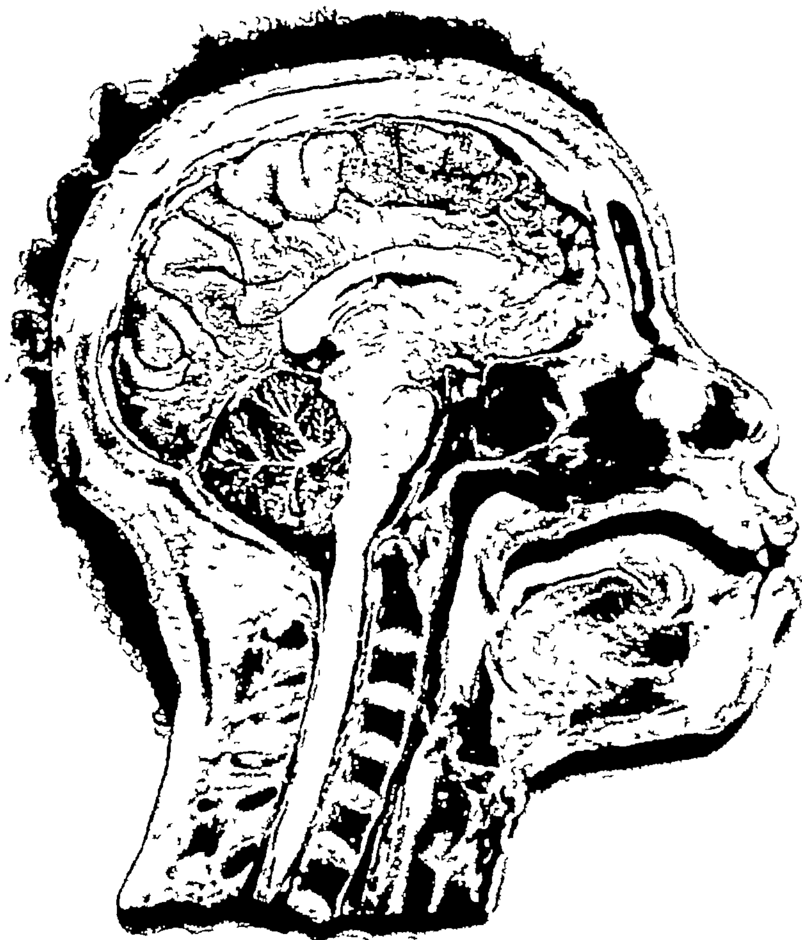


Fig 2 Median sagittal section of the head Note relation of brain to cranium

approximately under the line drawn on the surface of the skull from the angular process of the frontal bone to a point about a centimeter above the lambda

The central sulcus (Rolando) separates the frontal from the parietal lobe. It lies under a line drawn from the middle of the zygoma upward and backward to meet the sagittal

plane of the skull about 1 cm behind the midpoint between the glabella and external occipital protuberance

For practical purposes the cerebellum may be divided

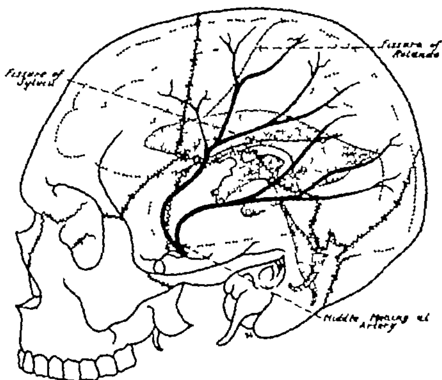


Fig. 3 Relationship of ventricular system to the brain and skull.

into two lateral lobes and a median lobe, or vermis The convolutions of the cerebellum are known as folia and are divided by parallel sulci

The Cerebral Ventricles

The lumen of the tube from which the central nervous system develops remains in the adult as the central canal of the spinal cord This is prolonged in the caudal portion of the medulla oblongata but soon becomes enlarged into a broad rhomboidal cavity the fourth ventricle (Fig 3) In the sagittal plane the fourth ventricle appears as a triangle

It has two lateral projections, the lateral recesses, which open into the subarachnoid space by means of the foramina of Lushka A third opening, the foramen of Magendie, is located in the median dorsal line at the caudal extremity of the fourth ventricle The fourth ventricle communicates with the third ventricle by means of the aqueduct of Sylvius which is bounded dorsally by the quadrigeminal plate and the posterior commissure and ventrally by the cerebral peduncles and the pons The third ventricle is a narrow cleft separating the optic thalami It communicates with the lateral ventricles by way of the foramen of Monro and the interventricular foramina Each lateral ventricle lies in the corresponding cerebral hemisphere and may be divided into a frontal horn, that portion anterior to the foramen of Monro, a body which extends from the foramen of Monro to a point opposite the splenium of the corpus callosum, a posterior horn which is an extension of the ventricular cavity into the occipital lobe, and a temporal horn extending into the temporal lobe

Chapter II

INTRACRANIAL PHYSIOLOGY

THE CEREBROSPINAL FLUID

There is still some controversy concerning the origin and function of the cerebrospinal fluid However for practical purposes we may consider that the cerebrospinal fluid is elaborated from the blood plasma by the choroid plexuses of the lateral third, and fourth ventricles The ependymal cells of the ventricles perivascular spaces of the brain, and blood vessels of the subarachnoid space also add to the supply of the cerebrospinal fluid. In many respects the cerebrospinal fluid behaves more like a dialysate than an active secretion The cerebrospinal fluid has the same osmotic tension as the blood plasma and alterations in the plasma content of chloride ions glucose and urea result in similar changes in the cerebrospinal fluid

The circulation of the cerebrospinal fluid has been termed by Cushing the "third circulation" This is not comparable to the circulation of the blood since the cerebrospinal fluid system does not have a central pump The movement of the fluid is dependent on differences of pressure in various parts of the system differences in chemical composition which promote diffusion on pulsations of the brain and alterations in the position of the body which permit the influence of gravity to act. The circulation of the cerebrospinal fluid does not have a constant directional course. Under normal conditions with the individual upright the fluid passes from the lateral ventricles through the foramen of Monro the third

ventricle, aqueduct of Sylvius, and fourth ventricle It enters the basilar cisterns of the subarachnoid space by passing through the lateral foramina of Lushka. The fluid then spreads upward around the brain stem and into the subarachnoid spaces over the hemispheres, from which it is absorbed into the venous system by means of the arachnoidal granulations (pacchionian bodies) Some of the fluid descends along the spinal subarachnoid space to be absorbed by the venous system of the spinal cord and spinal nerves. Absorption is facilitated by the fact that the hydrostatic pressure in the subarachnoid space is always greater than that in the dural sinuses.

The function of the cerebrospinal fluid is still partly shrouded in mystery The mechanical action of the fluid is self-evident It provides an efficient "shock absorber" for the entire central nervous system, shielding it from the jolts and jars of normal existence and protecting and minimizing the effects of more severe traumata The entire central nervous system is cushioned in a noncompressible fluid which absorbs and dissipates the usual and normal shocks and jolts of everyday activity

The large reservoirs of cerebrospinal fluid permit alterations in the volumes of the various components occupying the intracranial cavity For example, an increase in blood in the intracranial vessels, such as results from venous stasis in heart failure, is at the expense of the cerebrospinal fluid content Likewise, in cerebral edema, the brain enlarges due to an increase in intracellular fluid, reducing the amount of cerebrospinal fluid in the ventricular and subarachnoid spaces Conversely, in cerebral atrophy with reduction in brain volume, the cerebrospinal fluid spaces become enlarged

Theories as to additional functions of the cerebrospinal

fluid are conjectural Some writers consider the possibility that it is an avenue of escape for metabolic waste products of the nervous system or a means of distributing hormonal substances to various parts of the brain and spinal cord.

Normally with the individual in a horizontal position the cerebrospinal fluid is under a pressure of 70 to 160 mm of water In the sitting position the pressure measured at the lumbar level is about 200 mm higher due to the hydrostatic pressure of the column of fluid above the level measured whereas the pressure measured at the cisterna magna or ventricles may be zero or even less than atmospheric pressure Pressure of the cerebrospinal fluid closely parallels the intracranial venous pressure and changes in the latter are promptly reflected in the former This is demonstrated each time that a Queckenstedt test is performed. Compression of the veins in the neck interferes with venous return from the dural venous sinuses causing a rise in the intracranial venous pressure and concomitantly an increase in the cerebrospinal fluid pressure This rise in pressure promptly returns to normal when compression of the neck veins is discontinued. Anything which interferes with venous return from the cranium such as a constricting bandage around the neck, straining or coughing should be avoided when an increase in the intracranial cerebrospinal fluid pressure is undesirable

The volume of the intracranial cavity in an adult is practically constant although the large opening at the foramen magnum allows for very slight changes In the infant, as long as the fontanelles are open fluctuations in volume occur within narrow limits The practically constant intracranial volume in an adult is an important factor in the delicate balance between cerebrospinal fluid pressure and the volume of the cerebrospinal fluid, the blood the tissue fluids, and the

nervous tissue. Because of the nearly constant intracranial volume, changes in any of these components are reflected immediately in the others.

Following contusion of the brain there may be an increase in tissue fluids (edema). This of necessity causes a reduction in volume of cerebrospinal fluid or blood, or both. Reduction of blood volume is accompanied by some anoxemia and an accumulation of CO_2, both of which act as a stimulus to the vasomotor centers, provided they are in a condition to respond. Thus the blood pressure rises and the pulse slows. The increased blood pressure is transmitted by means of the capillary bed to the venous system, which elevates the venous pressure and coincidentally the intracranial pressure. This mechanism is seen in some cases of acute compression of the brain. In many cases, however, the injury to the brain has been so severe that the vasomotor center does not respond fully, if at all, and medullary compensation is interfered with.

Since the tension at which cerebrospinal fluid is filtered through the capillaries of the choroid plexuses is equal to the capillary pressure minus the osmotic tension of the nonpermeable elements of the blood plasma, intracranial pressure can be altered by changes in the osmotic tension of the blood. Because of this osmosis, hypertonic solutions, by vein or by mouth, produce a fall in cerebrospinal fluid pressure and, conversely, hypotonic solutions will produce a rise in the pressure.

Intracranial pressure may be altered by the administration of drugs. These act either by their influence on the osmotic pressure of the blood serum, or by producing a change in the intracranial venous pressures. Of the drugs commonly used, caffeine causes a rapid fall in the intracranial pressure. Caffeine and other xanthine derivatives pro-

duce a temporary alteration in the colloidal constituents of the blood plasma with a marked rise in osmotic tension in the blood plasma It is also probable that caffeine gives rise to a vasoconstriction in the cerebral circulation with a decrease in venous pressure Drugs, such as amyl nitrite which produces a vasodilatation of the cerebral vessels, cause a marked rise in intracranial pressure by increasing the blood volume in the intracranial cavity The usual inhalation anesthetics ordinarily increase intracranial pressure because of the asphyxia which attends their administration. During asphyxia there is always a marked rise in venous pressure with its accompanying rise in intracranial pressure As a matter of fact, any condition which interferes with the return flow of blood from the brain to the heart increases intracranial pressure.

BLOOD SUPPLY OF THE BRAIN

The brain derives its blood supply (Fig 4) from the two internal carotid and the basilar artery which unite to form the circle of Willis at the base of the brain The vertebral arteries pass along the lateral part of the spinal cord and medulla oblongata and unite at the posterior border of the pons to form the basilar artery The anterior inferior cerebellar arteries and the superior cerebellar arteries are derived from the basilar artery The posterior cerebral arteries are also branches of the basilar artery and furnish the blood supply to the under surfaces of the occipital and temporal lobes.

The internal carotid artery enters the skull through the foramen lacerum It traverses the cavernous sinus along its lateral wall, reaching the brain near the medial side of the temporal lobe where it bifurcates into the anterior and middle cerebral arteries The two anterior cerebral arteries are

joined by a small anterior communicating artery. The posterior communicating arteries connect each posterior cerebral artery with the internal carotid artery on the same side,



Fig 4 Visualization of the cerebral arteries in man by the injection of diodrast into the common carotid artery

thus completing the arterial circle at the base of the brain and providing a more or less efficient collateral circulation when one carotid or vertebral artery is occluded. The middle cerebral artery supplies the lateral part of the frontal lobe,

the insula, the upper surface of the temporal lobe most of the internal capsule and a greater part of convexity of the hemisphere The anterior cerebral artery supplies the mesial surface of the frontal and parietal lobes, most of the corpus callosum the optic chiasm and a portion of the anterior part of the internal capsule

The branches of the cerebral vessels which are known as main trunks run in the pia mater Contrary to popular impression they anastomose freely Small branches supply the cortex while other branches pass deeper to supply the white matter

The amount of blood within the intracranial cavity is dependent on the size of the vascular bed. The following factors influence this

1 *Vasomotor nerves* It has been definitely established that the cerebral arterioles have a sympathetic nerve supply Stimulation of the cervical sympathetics produces a brisk contraction of the pial arterioles and thus a diminution in the size of the vascular bed (blood volume) Recently a vasodilator mechanism of the cerebral arterioles has been demonstrated

2 *Chemical control* Increase in the CO_2 content of the blood produces local vasodilatation Histamine ergotamine tartrate and amyl nitrite also produce a dilatation of the cerebral arterioles

The sinuses of the dura mater form the main venous channels of the brain A description of the venous sinuses has been given in Chapter I The superior cerebral veins empty into the superior longitudinal sinus These veins drain the cortical and subcortical tissues of the mesial surface and part of the lateral surface of the cerebral hemispheres The inferior cerebral veins drain the under surface of the hemisphere, emptying into the middle portion of the lateral sinus

joined by a small anterior communicating artery The posterior communicating arteries connect each posterior cerebral artery with the internal carotid artery on the same side,



Fig 4 Visualization of the cerebral arteries in man by the injection of diodrast into the common carotid artery

thus completing the arterial circle at the base of the brain and providing a more or less efficient collateral circulation when one carotid or vertebral artery is occluded The middle cerebral artery supplies the lateral part of the frontal lobe,

the insula the upper surface of the temporal lobe most of the internal capsule and a greater part of convexity of the hemisphere The anterior cerebral artery supplies the mesial surface of the frontal and parietal lobes most of the corpus callosum the optic chiasm and a portion of the anterior part of the internal capsule

The branches of the cerebral vessels which are known as main trunks run in the pia mater Contrary to popular impression they anastomose freely Small branches supply the cortex while other branches pass deeper to supply the white matter

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The cerebral veins show many variations in their anatomical course and often the inferior cerebral veins may drain into the superior petrosal or cavernous sinus The great cerebral vein (vein of Galen) is made up of two large veins which drain the inner portions of the cerebral hemispheres These veins are formed by the confluence of veins from the anterior portion of the corpus callosum, the inferior surface of the frontal lobe, the area around the hypothalamus, the cerebral peduncle and the inferior horn of the lateral ventricle

FUNCTIONS OF THE CEREBRUM

The organization of the cerebral cortex into six cellular layers having distinctive differences in various areas indicates a functional specialization of the various parts of the brain The cellular structure (cytoarchitecture) of the cortex has been studied extensively by Meynert, Campbell, Brodman and more recently by von Economo and Koskinas The basis of classification is the distribution, size, and type of the neurones in each layer The white matter consists of the myelinated association paths which connect and integrate the various parts of the cortex with the rest of the central nervous system

On the basis of histological differences, and also by functional differences demonstrated by stimulation of the human brain at operation, we have now a good knowledge of the location of many functions The localization of other functions has been the result of careful study of postmortem and operative lesions

Frontal Lobe

The frontal lobe consists of that part of brain rostral to the fissure of Rolando, and above the fissure of Sylvius It

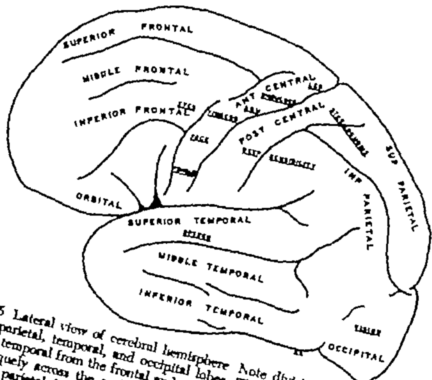


Fig. 5 Lateral view of cerebral hemisphere. Note division of cortex into frontal, parietal, temporal, and occipital lobes. The fissure of Sylvius separates the temporal from the frontal and parietal lobes. The fissure of Rolando runs obliquely across the surface of the hemisphere separating the frontal from the parietal lobe. The motor cortex lies in front of the fissure of Rolando, the sensory cortex behind it.

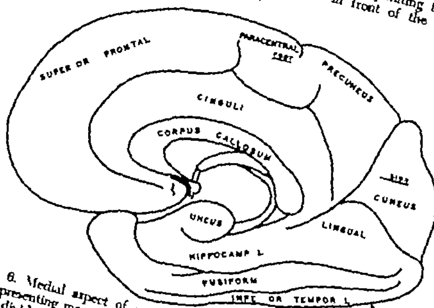


Fig. 6. Medial aspect of cerebral hemisphere. Note position of motor area representing movements of the foot. The basal surface of the temporal lobe is divided into inferior temporal, fusiform and hippocampal gyri and the uncus. The occipital lobe is separated from the parietal and temporal lobe by the calcarine fissure and the parieto-occipital fissure.

is divided into the motor, premotor, and prefrontal are
The motor area occupies the precentral gyrus on the late
surface of the hemisphere (Fig 5) It is the center for
litional control of somatic musculature Irritative lesions
the motor area produce Jacksonian motor convulsions. I
structive lesions result in loss of volitional control with
crease in muscle tone and hyperactivity of the tendon refle
(spastic paralysis) There is definite localization on the mo
cortex of movements of the various parts of the body T
representation on the motor cortex corresponds to the bo
inverted with the foot area high up nearest the midli
(Fig 6), and the representation of the face, jaws, and tong
low down near the Sylvian fissure. Stimulation of the mo
cortex produces movement of groups of muscles mediati
certain functions. The action of the motor area is modifi
by influences from many other cortical areas which ma
possible complex volitional responses The premotor ar
especially, correlates volitional movement.

The prefrontal cortex (the anterior portion of the fron
lobe) is especially concerned with psychic processes. I
juries of the frontal lobe cause slowness of cerebration, me
ory defect, emotional and behavior disturbances and in
tention With unilateral injuries the psychic disturbances m
be minimal or profound With bilateral frontal lobe injuri
the psychic disturbances are always profound, restlessne
incontinence, euphoria, disorientation, confusion, masturb
tion and voracious appetite often follow

Temporal Lobe

The temporal lobe is that area of the cerebrum inferi
to the Sylvian fissure, and merging posteriorly with th
parietal and occipital lobes The temporal lobes correla
auditory stimuli and probably also receive gustatory an
olfactory stimuli Lesions affecting the white matter of th

temporal lobe interfere with the radiations of the visual fibers as they make their sweep through the temporal lobe (Meyer s loop) on their way to the visual cortex in the occipital pole and cause homonymous field defects

Parietal Lobe

The parietal cortex is concerned essentially with somatic sensation Localization in the sensory cortex corresponds in general with that of the motor cortex Lesions of the sensory cortex produce loss of discriminatory sensation such as two point discrimination tactile localization and position sense Lesions in the supramarginal gyrus (posterior portion of the parietal lobe) result in the inability to appreciate the form of objects (astereognosis)

Occipital Lobe

The occipital cortex is the visual receptive center Each occipital cortex receives visual impulses from the temporal side of the retina of the ipsilateral eye and from the nasal retina of the contralateral eye Thus an occipital lobe lesion results in homonymous hemianopsia in the contralateral visual field (Fig 7)

A study of war injuries and lesions produced experimentally indicates that macular vision is represented in the posterior portion of the occipital poles while the cortex situated more anteriorly in the calcarine fissure serves peripheral vision Temporary cortical blindness due to contusion of both occipital lobes has been observed following cranio-cerebral injuries

SPEECH MECHANISM

In right handed individuals the left cerebral hemisphere controls the speech mechanism while in left handed persons this is a function of the right hemisphere Normal speech

depends on the integrity of several coordinated cortical areas. The areas especially concerned are the inferior frontal convolution, the superior temporal convolution, and the angular

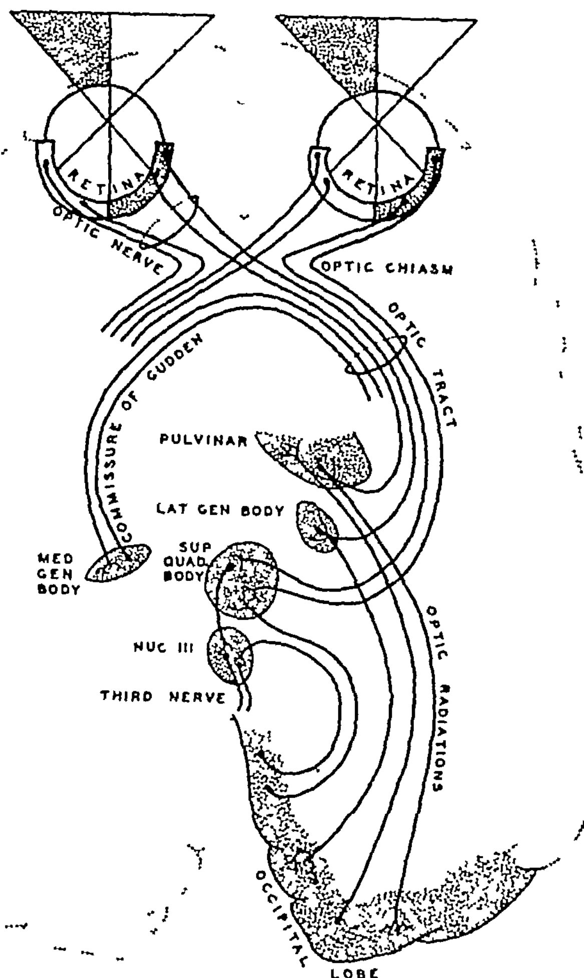


FIG 7 Diagram of the visual system The left visual field is represented *in the right occipital cortex, while the right field of vision is represented in* the left occipital cortex This is made possible by the decussation of the nerve fibers from the nasal half of each retina, so that each optic tract contains the fibers from the temporal retina of the same side and the fibers from the nasal retina of the opposite side

and supramarginal gyri of the parietal lobe Disturbances in the inferior frontal convolution give rise to dysfunction of

verbal or motor speech. A patient with a lesion localized in this area knows what he wants to say but the formulative motor mechanism is impaired. This is the so-called motor aphasia If he attempts to speak he may use the wrong word and is aware of the failure of his effort. He thereupon attempts to avoid the use of the wrong word and consequently the volume of speech is reduced.

With a lesion somewhat posterior and superior to the inferior frontal convolution (the motor writing center in the precentral gyrus) the patient suffers from agraphia, or inability to write

Alexia (inability to read) is caused by a lesion in the left angular gyrus A patient with such a lesion may not be able to name an object he sees but may be able to name it correctly if he feels it, provided the tactile sensory pathways are intact. If the gustatory or olfactory tracts are unimpaired he may be able to name the object by taste or smell

Anomia is the term given to the speech disturbance resulting from lesions in the posterior portion of the superior temporal convolution and the adjacent parietal tissue The patient is unable to call upon the correct word from the word memory center located in this region In this form of speech disruption the patient keeps repeating the wrong word (as differentiated from the patient with a lesion in the inferior frontal convolution) This is a sensory form of aphasia and he does not understand what he is saying The volume of speech may be increased In severe forms the patient will fluently emit a meaningless jargon

Because of the multiplicity of connecting tracts between the speech areas injuries that affect the speech mechanism rarely result in a single pure variety of aphasia Thus various combinations of sensory (receptive) and motor (expressive) aphasias result

Chapter III

THE MECHANICS AND PATHOLOGY OF HEAD INJURIES

THE MECHANICS OF SKULL FRACTURES AND BRAIN INJURIES

The anatomical structure and the elastic properties of the skull greatly influence the type of injury resulting from the application of a force to any part of the skull The lesions produced are also dependent on whether or not the head is stationary or in motion, in the latter case the velocity plays a major rôle

The energy of a blow by a blunt object traveling at a relatively slow speed and striking the stationary skull, most often produces the maximum damage to the bone and intracranial structures directly at the site of the impact The energy expended at the site of impact depends on the weight of the object and the square of its velocity With differences in the amount of energy, varying degrees of injury are produced Often the force is only great enough to deform the shape of the skull momentarily, the elastic properties of the skull resulting in a restoration of a normal contour without any dissolution of continuity The dura mater is practically never damaged in this type of injury because of its fibrous and elastic character The underlying brain, however, is almost always contused With forces of greater magnitude the deformation of the skull is greater The inner table, as a rule, fractures first and more extensively since it is less elastic than the outer table, and because of the curvature of the calvarium

With greater force both outer and inner tables may be fractured. If the object producing the trauma has a comparatively small surface, the energy will be expended over a relatively small area and if great enough, is more likely to produce a depression of the skull In addition the underlying brain may be contused The dura mater may or may not be torn, depending on the degree of the depression

The cortex underlying the fracture or depression will usually be covered by subpial hemorrhage, and in its substance one often finds a focal hemorrhage surrounded by small petechial hemorrhages

While most of the energy of a blow to the stationary head is expended at the site of impact, the elasticity of the skull results in the conversion of some of the energy into an indirect disruptive force This is brought about by a shortening of the diameter of the skull parallel to the axis of the applied violence, and a lengthening of the diameter at right angles to this axis Indirect disruptive force produces widespread petechial hemorrhages

Injuries sustained with the head in motion as in traffic accidents and falls, depend on the fundamental laws of motion When a head in forward motion is suddenly arrested by a massive stationary object, the greatest brain damage occurs at a point directly opposite the point of impact This is the so-called contrecoup injury (Fig 8) and its mechanism is as follows

The brain has many of the physical properties of fluids (which are inelastic) A localized increase in pressure will be transmitted as a wave at enormous velocity in all directions Part of the momentum of a moving head arrested by a massive stationary object is converted into such a localized pressure increase When the resulting wave reaches the opposite side, the skull at that point is still moving forward Thus the pres

the buttresses are weakened by numerous foramina for the passage of cranial nerves and blood vessels, thus adding to the susceptibility of these parts to fracture.

PATHOLOGY OF BRAIN INJURIES

Lesions present in the brain immediately following head trauma are the result of the applied violence plus the reaction of the brain and its vascular system

In the consideration of pathological changes in the brain following trauma it is wisest to disregard entirely the term "concussion" Those who prefer to retain this term (which is a poor one even when employed clinically), should restrict its use to "indicate an essentially transient state due to head injury which is of instantaneous onset, manifests widespread symptoms of a purely paralytic kind, does not as such comprise any evidence of structural cerebral injury, and is always followed by amnesia for the actual moment of the accident" (Trotter) Few persons die of "concussion" The anatomical changes, if any exist, are therefore practically unknown

Cerebral Edema

The occurrence of edema of the brain following head trauma is admitted by most students of the subject Its degree and clinical importance have been the cause of much controversy Examinations of human postmortem material, and that obtained from experimental studies have not yielded consistent results Nevertheless, it is impossible to deny the frequent, though not constant, finding of increased fluid in the brain and meninges of persons dying several hours to two or three days after a severe head injury Many blood vessels rupture following trauma, and others are occluded by thrombi, thus large areas of brain tissue suffer from anoxemia. Tissues deprived of oxygen take up fluid and swell (Cannon).

The brain becomes edematous and venous channels are compressed with a consequent elevation of venous pressure This impedes the passage of cerebrospinal fluid from the sub arachnoid spaces into the venous system In addition blood in the subarachnoid spaces mechanically retards absorption Both of these factors result in an increased amount of free fluid in the cerebral sulci and ventricles Pathological studies made by Rand and Courville of many brains from patients dying of acute head injuries showed a definite increase in the fluid content The increased fluid content was due to an excessive amount in the subarachnoid spaces and ventricles the "free fluid" Histological preparations showed enlargement and vacuolization of the cells of the choroid plexuses, edema of the ependymal cells and subependymal tissues and increase in size of the pericellular spaces of the brain substance itself—"bound fluid"

Cerebral Contusion (Bruising of the Brain)

Contusion of the brain occurs to a greater or less degree in most head injuries except in the mildest ones With direct blows to the stationary head contusion is most marked at the site of impact With the head in motion the contusion is more widespread but the greatest contusion is found on the under surfaces of the frontal lobes and at the tips of the temporal lobes Contusions of the brain vary in severity from those which are superficial, and over which the meninges remain intact to those in which the brain is almost pulpified and the meninges extensively lacerated The latter type of contusion is frequently associated with more or less laceration of the brain Superficial contusions are those in which numerous petechial hemorrhages into the brain tissue have occurred and the cytoarchitectonics have been mechanically disrupted In the more severe contusions, the brain is infiltrated

with blood for a distance of 2 to 4 cm The capillaries and smaller arterioles and venules are dilated and the coats of the vessels may be disrupted with hemorrhage into the adventitia.

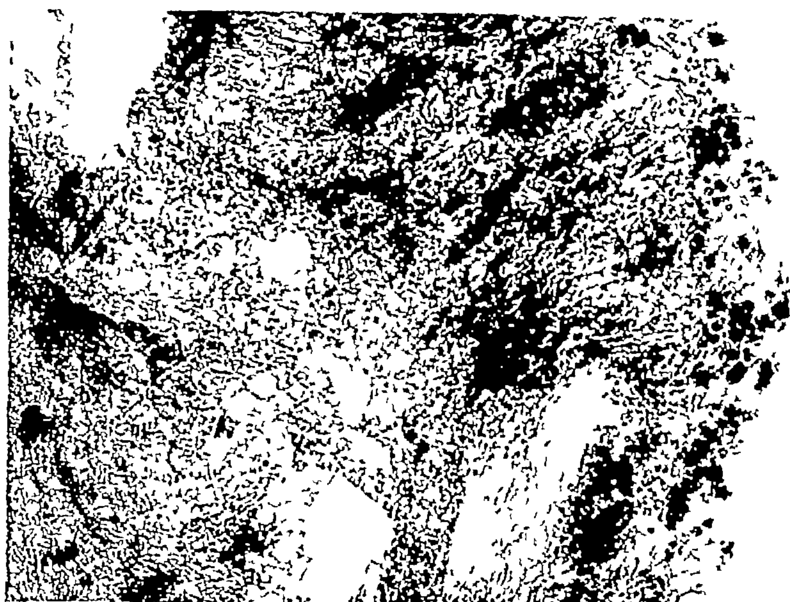


FIG 10 Deposition of fatty granules in an area of contused brain Fat stain, × 120

Thrombi are found in the lumen of the vessels On microscopic examination the cerebral tissue and its fissures are filled with hemorrhagic material and broken-down blood pigment The cortex stains poorly and the cytoarchitectonics are destroyed The ganglion cells show all types of pathological changes Marked changes are found in the glial elements at an early stage. The processes of the astrocytes become swollen and granular At the end of three or four days, amitotic division of astrocytes is found At this time the glial fibrils, previously present in the astrocytes, become invisible After all division is completed, new fibrils are laid down. The microglia in the injured area are spurred into activity. There is at first a great influx of microglial cells into the injured region Within twelve hours the processes of the

microglia are retracted and thickened, and after twenty four hours many ameboid forms showing active phagocytosis are found In many of them ingested blood cells are seen The digestion of blood elements and particles of traumatized cerebral tissue results in the production of lipoid substances (Fig 10) These fat laden phagocytic cells are the typical "fat granule cells" or "gitter cells"

Cerebral Laceration

Laceration of the brain (Fig 11), similar to contusion may occur at the site of impact, or in areas far removed The location of the lesion follows definite physical laws which have already been described. Direct lacerations correspond to the site of fracture Lacerations are produced when the force of the injury is great enough to exceed the limits of cohesion of the cerebral tissue Contrecoup lacerations are due to violent contact of the brain with the side of the skull opposite the site of the application of the force Lacerations are also frequently found on the under surface of the frontal lobe and tips of the temporal lobe With severe injuries the lacerations may extend into the ventricle, with extensive hemorrhage

The microscopic changes are similar to those found in contusion but they are more profound. In addition to the glial reaction there is marked mesenchymal proliferation In the healing of these lacerations (Fig 12) the fibroblastic elements take an active part. Numerous capillaries develop in the newly formed tissues which later produce a fibrous scar between the dura and pia mater From this scar strands of fibrous tissue project into the underlying cerebrum Surrounding the fibrous tissue a wide zone of fibrillary astrocytes is found

When the laceration is caused by a bullet the reaction of

the brain differs somewhat In general, the edema is much greater than that produced by other injuries The track of the missile contains necrotic and hemorrhagic brain tissue



Fig 11 Traumatic laceration of the left frontal lobe

For about a centimeter around the track there is marked edema and hemorrhagic infiltration In the next zone, numerous ring hemorrhages surround the small blood vessels The pathological changes gradually decrease toward the periphery of the track.

Lacerated brain is at first bright red, due to infiltration with blood Later, as the blood and blood pigments are phagocytosed, the area becomes brownish-red Finally, a scar composed of a fine fibrous meshwork, containing calcified

ganglion cells is formed—the so-called *plaque jaune* Meningocerebral adhesions surround the old laceration. Lacerations which extend into the ventricle form cavities containing



FIG. 12. Healing laceration of frontotemporal region. This patient died of bronchopneumonia three weeks after a severe head injury

cerebrospinal fluid These cavities are traumatic porencephalic cysts and their lining is composed of glial fibrils

Traumatic Cerebral Hemorrhage

Intracranial hemorrhage accompanies a large proportion of all moderately severe and severe head injuries as attested by the frequency with which blood is found in the spinal fluid when lumbar puncture is performed Subpial and subarachnoid bleeding is part of the pathological picture in all cases of cerebral contusion and laceration Petechial hemorrhages and

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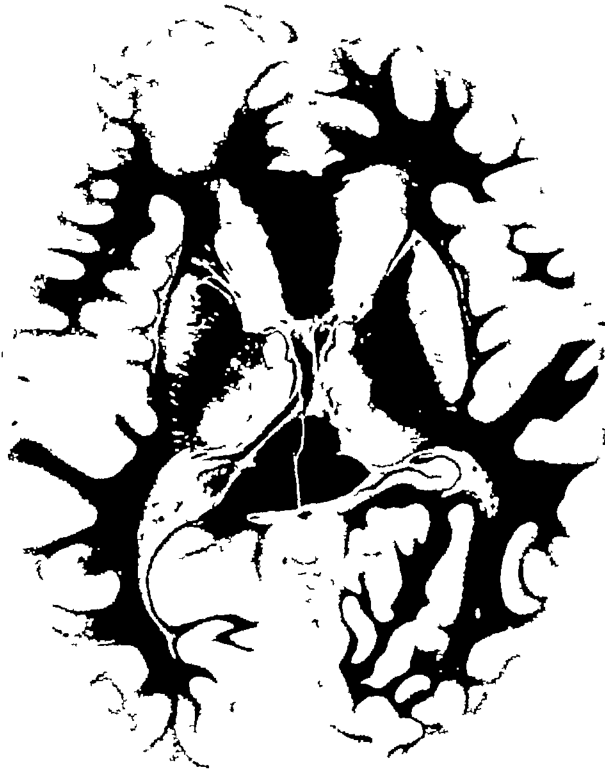


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larger focal intracerebral hemorrhages (Fig 13) also occur frequently. Gross hemorrhages in the pons and medulla oblongata are found in fractures of the base, especially when



Fig 13 Large focal intracerebral hemorrhage due to trauma Note the smaller petechial hemorrhages in the white matter of the opposite hemisphere

the middle or posterior fossae are involved When the leptomeninges are torn, blood is usually found in the subdural space These subdural collections of blood are always associated with subarachnoid bleeding, the blood entering the subdural space through tears in the arachnoid membrane Although extradural hemorrhages are infrequently diag-

nosed clinically they are found in approximately 10 per cent of all patients who die of head injury

Traumatic Subpial and Subarachnoid Hemorrhages

The cortical veins are thin walled and inelastic Their location in shallow grooves on the surface of the brain makes them particularly vulnerable to injury They are often torn without laceration of the brain itself Subpial hemorrhage is caused by tearing of cortical vessels When the pia mater tears subpial blood escapes in the subarachnoid space and is diffused through the cerebrospinal fluid passageways With the tearing of small cortical vessels, the pia mater remaining intact many small subpial hemorrhages are formed.

The presence of blood and the products of its disintegration in the cerebral sulci and cisterns increase the osmotic pressure of the cerebrospinal fluid This impedes the absorption of the cerebrospinal fluid into the venous system In addition there is mechanical interference with the free flow of cerebrospinal fluid due to the clogging of the sulci and cisterns with red blood cells

Petechial Hemorrhages

Small pinpoint hemorrhages in the white matter (Fig 14) and in the basal ganglia are found in the brains of most patients who die of head injury It is probable that they occur in many of the patients who recover from moderately severe and severe head injuries These hemorrhages are found in the perivascular spaces and are often known as ring hemorrhages Cassasa believed they were due to a sudden over filling of the perivascular spaces (Virchow Robin spaces) with cerebrospinal fluid rupturing the vessel wall These small punctate hemorrhages are followed by coalescent areas of demyelinization in the white matter The destructive proc

ess is followed by a marked reaction of the microglia and astrocytes. In the adjacent cortex there is often distortion of the cytoarchitectonics with disintegration and ischemic

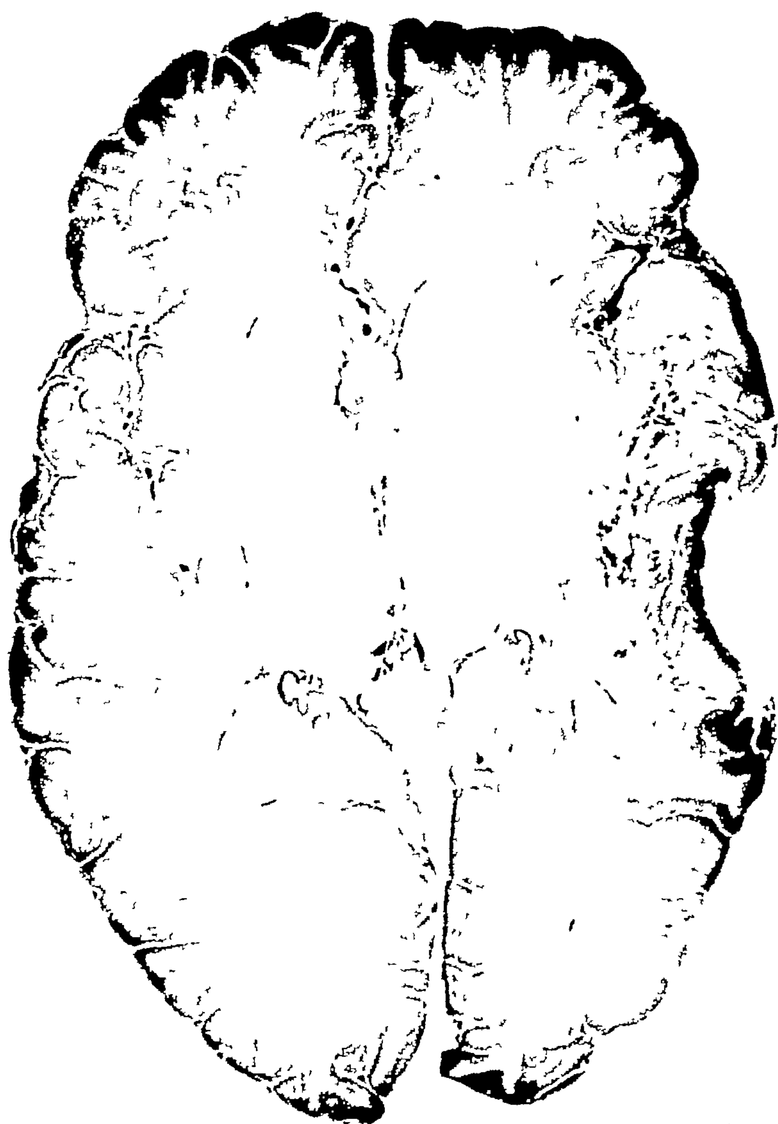


Fig 14 Petechial hemorrhages in the white matter of the brain of a patient who died a few days after a severe head injury

changes in the ganglion cells These cortical areas stain poorly

Medullary and Pontine Hemorrhages

Traumatic hemorrhages into the pons and medulla oblon gata occur in about 7 per cent of the patients who die of head injury They occur most frequently in fractures of the pos terior and middle fossae and are often symmetrical The blood supply of the pons is derived from branches of the basilar artery which leave the main trunk at right angles With stretching of the cerebrum from the brain stem these vessels have a tendency to rupture The symmetry of the lesions is explained by the fact that the blood supply to the pons and the medulla oblongata is symmetrical and that the energy reaching the brain stem which is at a relatively remote distance from the point of impact, is distributed in such a manner that symmetrical branches are equally affected

Chapter IV

METHODS OF EXAMINATION

A patient who has suffered a head injury, when first seen, is often comatose or uncooperative The value of an accurate history is self-evident, though often unobtainable since many patients are brought to the hospital following street accidents In such cases reliable witnesses or accurate information are often unavailable When a history cannot be obtained, but there is external evidence of an injury to the head, the condition should be considered as due to trauma until proved otherwise

If obtainable, data concerning the age, sex, race and occupation of the patient should be recorded on the chart. The type of accident and the time when it occurred should be learned if possible Also details of past illnesses, particularly heart disease, convulsive seizures, diabetes, hypertension, drug or alcohol addiction, which may have caused the patient to fall and suffer a head injury

The first medical observer should make a rapid appraisal of the condition of the patient and note his findings carefully. The state of consciousness, color, pulse rate, temperature and blood pressure are most important The escape of blood, spinal fluid or brain tissue from the orifices, and the presence of injuries of other organs or of the extremities must be looked for The odor of the breath may aid in detecting diabetic coma, uremia, or alcoholism

Often, the condition of the patient permits only a brief neurological examination, yet a careful appraisal of the nerv-

ous system is necessary since treatment is greatly influenced by alterations in the nervous structure

The preliminary examination of the central nervous system includes a brief mental examination In this connection it is most important to note in the record in unmistakable terms the exact state of consciousness The terms "coma" "stupor" and "drowsiness" are often misused. Strictly speaking coma should be used to denote a state of complete loss of consciousness in which the patient cannot be aroused even by the most painful stimuli. Stupor indicates partial loss of consciousness A patient in stupor reacts to painful stimuli such as pressure over the supra-orbital nerve or pin pricking Drowsiness signifies a state in which the patient falls asleep if left alone If the patient is conscious it is important to determine whether or not he is oriented as to time place and person Even in a brief interview memory can be tested Many patients with head injuries have complete amnesia for the accident and the events immediately preceding it

The emotional attitude should be ascertained, especially where the head injury may have been self inflicted. Extreme restlessness and delirium are usually indicative of frontal lobe damage The scalp and skull must be carefully examined Small lacerations and bruises are often hidden by hair especially in women and their true extent can be determined only after the scalp has been clipped and shaved Irregularities of the skull, visible fractures depressions pulsations escape of blood cerebrospinal fluid or brain tissue from an open head wound should be noted Auscultation and percussion of the head are often neglected yet they may occasionally be of extreme diagnostic importance

In fractures through the sphenoidal fissure the carotid artery may be torn as it passes through the cavernous sinus with the formation of an arteriovenous aneurysm The pa

tient may voluntarily complain of a "noise" in the he
this fact may be brought out only by careful question
a stethoscope is placed in the frontal or temporal re
swishing bruit is heard, which can be obliterated by co
sion of the carotid artery on that side

There is often some tenderness on percussion over a
ture of the vault, and definite dullness on the injured

Smell can rarely be tested at the first examination, si
this time the patient is usually either unconscious, c
cooperative When the condition of the patient permits,
should be tested, since the olfactory nerve, tract, or bu
injured occasionally, with complete loss, or diminut
smell.

The next important procedure is an examination c
pupils and the pupillary light reflex It is important,
fore, that no drug which influences the size or reaction
pupil should be given. Bilaterally dilated pupils which c
react to light, are found in patients with very severe
injuries, and commonly indicate impending death A
laterally dilated and fixed pupil is frequently found o
side of an extradural hemorrhage, and occasionally o
side of a subdural hemorrhage or hematoma Unil
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The immediate examination of the visual system in pa
with acute head injuries is usually limited to testing the p
lary reactions and observation of the fundi The pat

condition most often precludes the determination of the visual acuity and visual fields though both should be done before the patient s discharge

For the fundus examination a mydriatic should not be employed since any interference with pupillary reactions may obscure important diagnostic data.

The margins of the optic discs are normally well defined, though a slight blurring of the nasal margins is within physiological limits Swelling of the disc is rarely observed early in acute head injuries though the retinal vessels are frequently congested Hemorrhages tears and detachment of the retina may occur when the head injury is associated with direct trauma to the eyeball Patients with subdural hematomas may have slight swelling of the optic discs though in a few cases marked swelling has been noted.

Paralysis of the extraocular muscles is often overlooked in the early stage of an acute head injury especially during the period of unconsciousness and poor cooperation. Complete paralysis of the oculomotor or abducens nerves is observed infrequently The abducens because of its long course along the base of the skull is the more vulnerable of the two Oculomotor palsy is observed occasionally in patients with middle meningeal hemorrhage Slight degrees of diplopia are best elicited with a red glass

Disturbances of fusion convergence and accommodation are frequently seen after head injuries and are most often discovered during convalescence when the patient attempts to read The mechanism controlling conjugate movements of the eyes (lateral upward and downward gaze) is an intricate one Cerebral centers controlling conjugate eye movements are located in the second and third frontal convolutions and also in the parietal region An irritative lesion produces forced deviation of the eyes to the opposite side

tient may voluntarily complain of a "noise" in the head, or this fact may be brought out only by careful questioning. If a stethoscope is placed in the frontal or temporal region a swishing bruit is heard, which can be obliterated by compression of the carotid artery on that side

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Smell can rarely be tested at the first examination, since at this time the patient is usually either unconscious, or uncooperative When the condition of the patient permits, smell should be tested, since the olfactory nerve, tract, or bulb are injured occasionally, with complete loss, or diminution of smell

The next important procedure is an examination of the pupils and the pupillary light reflex It is important, therefore, that no drug which influences the size or reaction of the pupil should be given Bilaterally dilated pupils which do not react to light, are found in patients with very severe head injuries, and commonly indicate impending death A unilaterally dilated and fixed pupil is frequently found on the side of an extradural hemorrhage, and occasionally on the side of a subdural hemorrhage or hematoma Unilateral mydriasis also occurs following direct trauma to the eyeball, with or without concomitant intracranial injury. Miotic, fixed pupils are occasionally observed after head injuries If tabes dorsalis, or other syphilitic disease of the central nervous system can be eliminated, miotic fixed pupils indicate injury or hemorrhage in the midbrain Horner's syndrome (miosis, enophthalmia and ptosis) indicates damage to the sympathetic pathways, either in the brain stem or in the neck

The immediate examination of the visual system in patients with acute head injuries is usually limited to testing the pupillary reactions and observation of the fundi The patient's

condition most often precludes the determination of the visual acuity and visual fields, though both should be done before the patient s discharge

For the fundus examination a mydriatic should not be employed since any interference with pupillary reactions may obscure important diagnostic data

The margins of the optic discs are normally well defined though a slight blurring of the nasal margins is within phys iological limits Swelling of the disc is rarely observed early in acute head injuries though the retinal vessels are frequently congested Hemorrhages tears and detachment of the retina may occur when the head injury is associated with direct trauma to the eyeball Patients with subdural hema tomas may have slight swelling of the optic discs though in a few cases marked swelling has been noted.

Paralysis of the extraocular muscles is often overlooked in the early stage of an acute head injury especially during the period of unconsciousness and poor cooperation Complete paralysis of the oculomotor or abducens nerves is observed infrequently The abducens, because of its long course along the base of the skull, is the more vulnerable of the two Oculomotor palsy is observed occasionally in patients with middle meningeal hemorrhage Slight degrees of diplopia are best elicited with a red glass

Disturbances of fusion convergence and accommodation are frequently seen after head injuries and are most often discovered during convalescence when the patient attempts to read. The mechanism controlling conjugate movements of the eyes (lateral, upward and downward gaze) is an intricate one Cerebral centers controlling conjugate eye movements are located in the second and third frontal con volutions, and also in the parietal region An irritative lesion produces forced deviation of the eyes to the opposite side

whereas with a destructive lesion there is paralysis of conjugate movements to the opposite side. This can often be of value in determining whether a traumatic lesion of the brain is intracerebral or extracerebral. Assuming that most extracerebral lesions (such as subdural and extradural clots or fluid collections) are irritative, these conditions would show forced deviation of the eyes toward the side of the hemiplegia With an intracerebral (supposedly destructive) lesion the patient would be unable to look toward the hemiplegic side, and there might even be transient deviation of the eyes toward the side of the lesion

Traumatic lesions of the midbrain may also be associated with disturbances of conjugate movements of the eyes With a lesion in this location, however, there are pupillary changes, nuclear ocular palsies, and bilateral pyramidal tract signs

The trigeminal nerve is not often involved in head injuries Disturbances in sensation of the face, corneal reflexes, and motor power of the jaw are easily tested It should be remembered that in a motor trigeminal weakness, the jaw deviates to the side of the lesion due to the action of the opposite intact pterygoid muscles

Unilateral peripheral facial paralysis is a frequent complication of head injuries In most cases it comes on twenty-four to seventy-two hours after the accident, and is due to bleeding or edema around the sheath of the nerve in the facial canal Taste of the anterior two-thirds of the tongue is affected, since the nerve is usually injured proximal to the point where the chorda tympani leaves it to join the lingual branch of the mandibular nerve

The auditory nerve is also frequently involved, especially in basal fractures which traverse the petrous bone. In some cases bilateral nerve deafness follows basal skull fractures The hearing defect at first may be of a combined conduction

and nerve type, since there is often bleeding into the middle ear and rupture of the tympanic membrane

Hearing is most conveniently tested by noting the distance from either ear at which the patient is able to detect the tick of an ordinary pocket watch The difference between air con duction and bone conduction may be compared by first placing the handle of a C-256 tuning fork on the mastoid process until the patient can no longer hear the sound The tuning fork is then placed with its branches close to the auricle Normally air conduction exceeds bone conduction If any hearing defect exists, when the patient is convalescent more careful tests should be made using the audiometer

The vestibular nerve and labyrinth are often injured in basal skull fractures Dizziness on changing position, and spontaneous nystagmus indicate a lesion of the labyrinth vestibular nerve or its pathways Nystagmus is designated by its quick component and is further classified as to direction—horizontal rotatory or upward Rhythmical nystagmus (in which the oscillations are of equal speed and amplitude) is often congenital

When any injury to the vestibular nerve is suspected caloric tests should be made before the patient is discharged. A simple method is to irrigate the external canal with cold water after examining it to make sure that the water reaches the drum The suspected abnormal ear should be tested first Normally the reaction begins in about thirty seconds and consists of nystagmus away from the irrigated ear, dizzi ness falling and past pointing toward the side of the irri gated ear This reaction lasts about two minutes Caloric tests should not be done if the tympanic membrane has a per foration

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palate at rest and in phonation, gag reflex, swallowing, and cough reflex should be noted The power of the trapezius and sternocleidomastoid muscles, and the position of the tongue when protruded complete the examination of the cranial nerves

The extremities should be examined as to position, deformities, motor power, tremors or other involuntary movements In hemiparesis the lower extremity is held in a position of eversion, but in an unconscious patient this must be differentiated from a fracture of the neck of the femur The deep reflexes are often absent immediately after a severe head injury. They usually return when the patient has recovered from shock Hyperactive deep reflexes are frequently found in severe head injuries, and probably arise from edema or bleeding around the brain stem A bilateral Babinski sign may be present for the first few days after a severe head injury As a rule, the plantar response returns to normal within a few days in a patient who recovers A Babinski sign may occasionally persist on one side for several weeks with very little motor weakness An injury of the spinal cord may not be discovered for several days when there is an associated head injury. Retention of urine and bilateral abnormal plantar responses should lead one to suspect damage to the spinal cord, even in a patient who remains comatose for several days

Weakness of both lower extremities with a depressed fracture of the convexity of the skull results from occlusion of the superior longitudinal sinus and interference with venous return from both hemispheres

Sensory examination is, of course, impossible in a comatose patient. Injured persons who are stuporous or drowsy respond to painful stimuli, and it is important in such cases to determine by their reaction to painful stimuli whether

there is any alteration in their sensory status When the pa tient is able to cooperate a careful sensory status should be recorded.

Evidence of meningeal irritation should be looked for Neck rigidity and Kernig s sign when positive following a head injury usually indicate the presence of blood in the cerebrospinal fluid When these signs appear after a lapse of forty-eight hours or more meningitis must be ruled out

LABORATORY AIDS

When a patient is comatose and there are no available data, laboratory aid may be needed to arrive at a correct diag nosis Diabetic coma is nearly always easily recognized. The urine should be examined as soon as possible, obtaining a specimen by catheter if necessary Blood chemistry and serological tests should be made routinely in every case of head injury Uremia is usually easily recognized by the uriniferous odor of the breath high blood pressure urine and blood chemistry findings

Meningitis and encephalitis can be ruled out by a lumbar puncture The place of lumbar puncture in the diagnosis and treatment of head injuries will be discussed more fully in a later chapter It should be done early only when doubt exists as to the presence of a head injury The indications are coma without available history stiff neck, or fever In most head injuries severe enough to produce lasting coma, blood is found in the spinal fluid All the specimens are equally col ored, and when the cerebrospinal fluid is examined freshly many of the cells will be found to be crenated. The pressure of the cerebrospinal fluid should always be measured, prefer ably with a water manometer If high the pressure must not be reduced too rapidly and the final pressure must not be reduced more than one-half of the initial one

In meningitis the cerebrospinal fluid is turbid or cloudy and contains many polymorphonuclear cells Protein is increased and sugar is absent A smear and culture disclose organisms In encephalitis the fluid may be clear, or slightly cloudy The cell count may be normal, or as many as 200 to 1000 cells, mostly lymphocytes, may be found Sugar is present, and may be increased above normal. Tuberculous meningitis in an adult may baffle the examiner for some time. In this condition, the cerebrospinal fluid is cloudy, sugar is reduced, the chlorides greatly diminished and the tryptophane and Levinson tests are positive

A moderate leucocytosis usually follows a head injury. If the patient presents signs of severe secondary anemia due to bleeding from an extensive scalp laceration, his blood should be typed for transfusion. This is especially important if any operative procedure is contemplated

X-RAY EXAMINATION

X-ray examination of the skull should be delayed until the condition of the patient permits Films taken with a portable apparatus, and those taken in uncooperative individuals are usually valueless, and the results of an x-ray examination do *not often alter the method of treatment* decided upon after careful clinical examination and *observation. Roentgenography* of the skull is an urgent procedure *only* if a compound or depressed skull fracture requiring immediate surgical intervention is thought to be present. When an extradural hemorrhage is suspected, x-ray evidence of a fracture crossing the channels for the middle meningeal vessels is of diagnostic importance Needless to say, patients with severe head injuries and in shock require immediate care. The x-ray examination is of secondary importance, and in many cases may wisely be delayed for several days However, no patient

who has had a head injury should be discharged before satisfactory roentgenograms of the skull have been obtained A compound depressed skull fracture may occasionally be sustained with slight or no disturbance of consciousness and such patients are often sent home without x ray studies after first aid has been administered

A minimal x ray examination consists of a lateral view of each side and anteroposterior view and a film of the base If the findings are doubtful, stereoscopic examination is indicated We have found the Bucky diaphragm helpful in obtaining satisfactory detail in skull films Many fractures of the skull, proved by operation or autopsy are not demonstrated by the x ray especially if the fracture is limited to the base

The distinction as to whether a fracture is of the vault or base is not of great importance since many fractures of the vault extend into the base It is now well recognized that the damage to the bony structure is of secondary importance compared to the injury to the brain, its membranes and blood vessels The extent of damage to the skull is however some indication of the severity of the blow and thus becomes an important clinical factor Also the presence or absence of a fracture is important when an open wound is present when the fragments are depressed, when the fracture extends into the nasal sinuses or crosses the groove of the middle meningeal artery or any of its branches

Simple linear fractures of the vault (Fig 15) are usually easily recognized. They can be differentiated from vessel markings by their direction and sharp outlines They should be carefully searched for and reported since legal opinion still places undue weight on the presence or absence of a fracture in the roentgenogram

Simple depressed skull fractures (Figs 16, 17, 18, 19) are not always revealed in flat films, but may require stereoscopic views for their demonstration. Many errors are made clini-



Fig 15 Linear skull fracture

cally even by the experienced Effused blood under the periosteum feels at the periphery like a gentle, sloping, thick mound with a sharp edge, while the center of the blood-soaked tis-

FIG. 16. Depressed skull fracture.



FIG. 17 Comminuted, slightly depressed skull fracture. This patient was struck over the head with a hammer

sues simulates a depression in the skull. When a depression does exist, it is nearly always more extensive than an x-ray film would lead one to believe.

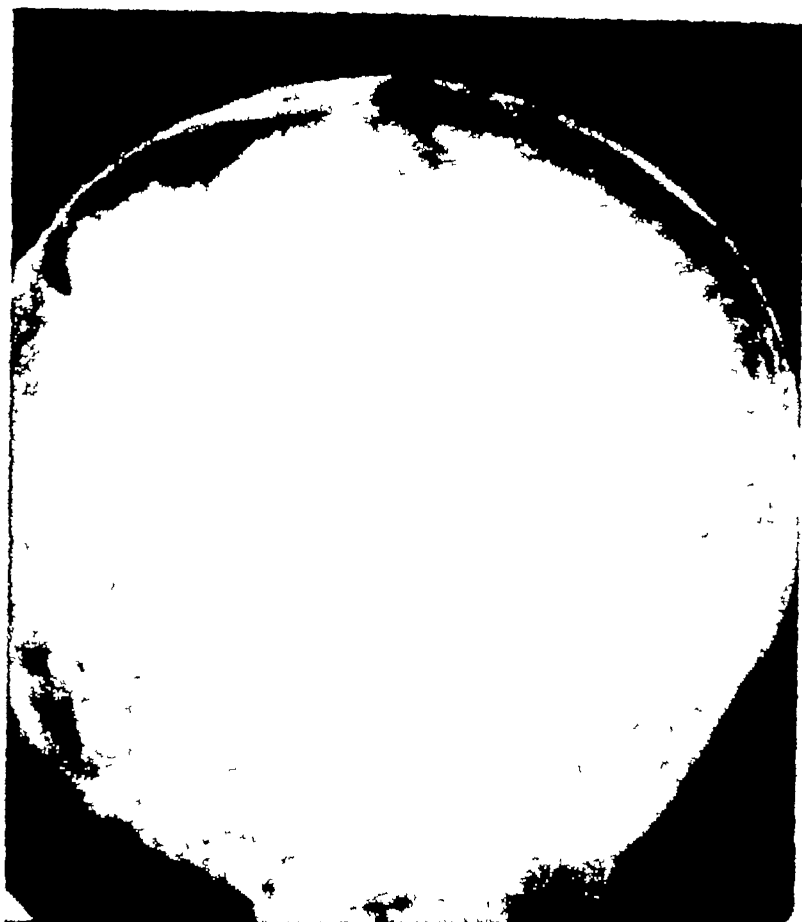


FIG. 18 Extensive depressed skull fracture

An extensive depression of the skull may be present without evident external wound, especially in infancy and childhood, when the scalp is thin and elastic, and when little or no hemorrhage occurs into the scalp and periosteum (Figs. 20 and 21).

Fractures of the base of the skull (Fig 22) are not always easily demonstrated in x-ray films They should be suspected



FIG. 19 Depressed skull fracture on left posterior parietal region. T patient had a right hemiplegia and aphasia.



FIG 20. X-ray film of skull showing depressed fragment of bone turned in at right angles. There was no evident external wound until the scalp was shaved, when a small bruise was discovered



FIG 21. Postoperative x-ray of the same case shown in Figure 20. The depressed bone fragment was used to fill in the defect. In spite of an intact scalp the dura and brain were lacerated. Note the silver clips used in controlling hemorrhage from the traumatized area.

when subcutaneous effusion of blood appears in a few hours, or even a few days following the trauma Effusions in the region of the orbit signify a basal fracture of the frontal fossa



FIG. 22. Basal skull fracture extending into the foramen magnum Film taken in the "Towne" or cerebellar position

while effusions over the mastoid tip (Battle's sign) indicate a fracture of the middle or posterior fossa.



Fig 20 X-ray film of skull showing depressed fragment of bone turned in at right angles There was no evident external wound until the scalp was shaved, when a small bruise was discovered



Fig 21 Postoperative x-ray of the same case shown in Figure 20 The depressed bone fragment was used to fill in the defect In spite of an intact scalp the dura and brain were lacerated Note the silver clips used in controlling hemorrhage from the traumatized area

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FIG. 22. Basal skull fracture extending into the foramen *magnum* Film taken in the "Towne" or cerebellar position.

while effusions over the mastoid tip (Battle s sign) indicate a fracture of the middle or posterior fossa

Fractures of the base have a predilection for certain areas, such as those containing foramina or nerve canals where the anatomical structure is weak Fractures often traverse the

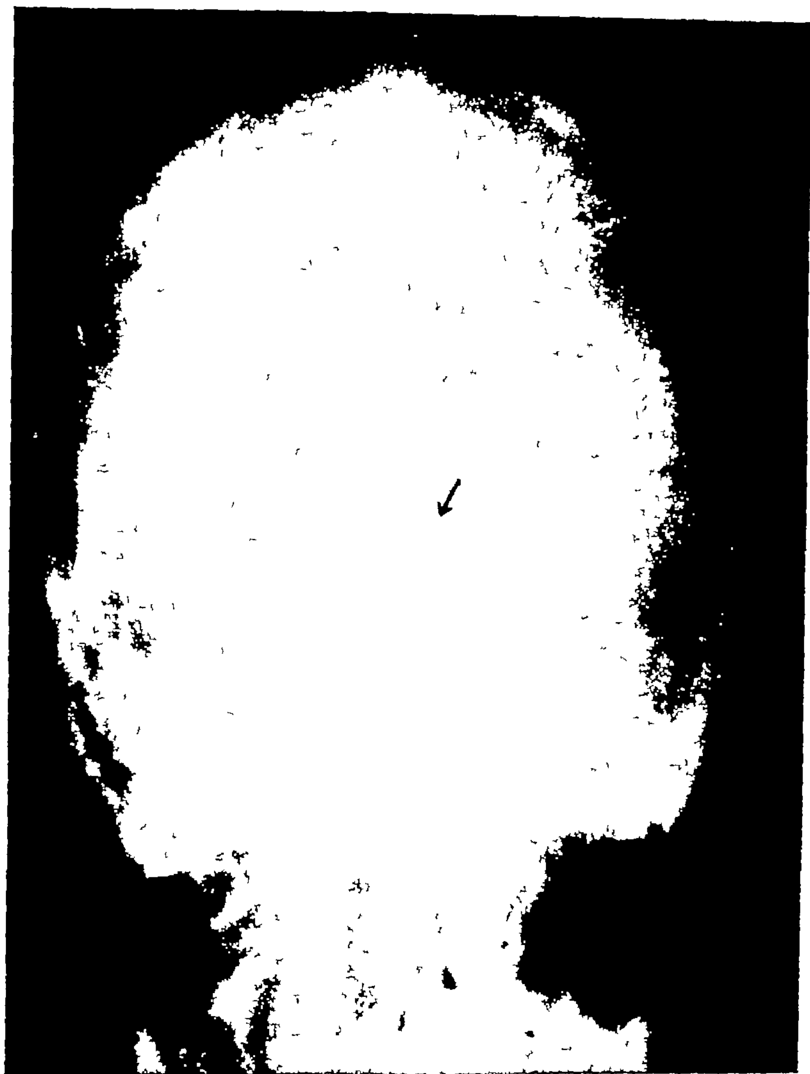


Fig 23 Pineal gland displaced to the right by left-sided subdural hematoma

cribriform plate which is perforated by the many filaments of the olfactory nerve The petrous bone is weakened by the three chambers of the auditory apparatus, and the canals of the seventh and eighth nerves, and is also subject to frequent fracture

The pineal gland is calcified in about 60 per cent of adult skulls It is normally in the midline and occupies a definite position in relation to the anteroposterior and vertical diam

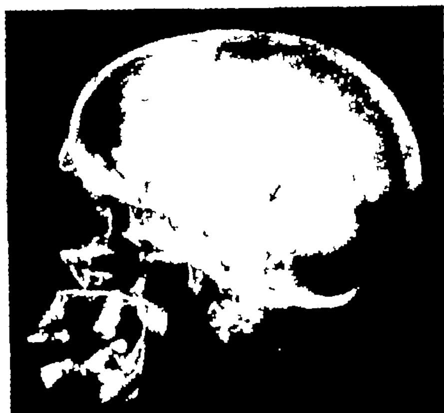


FIG. 24 Backward and downward displacement of the pineal gland in frontal subdural hematoma.

eters of the skull A shift to the left or right of the midline can be easily seen and indicates a space-containing lesion The pineal gland may be displaced by a traumatic subdural hematoma (Fig 23) To determine its displacement in the anteroposterior and vertical diameters of the skull (Fig 24) its position must be plotted according to the method of Vastine and Kinney

Pneumo-encephalography and ventriculography are valuable adjuncts to the diagnosis of traumatic intracranial le-

sions Encephalography is of especial aid in the study of post-traumatic states, while ventriculography is sometimes employed to localize a traumatic subdural hematoma (Fig.

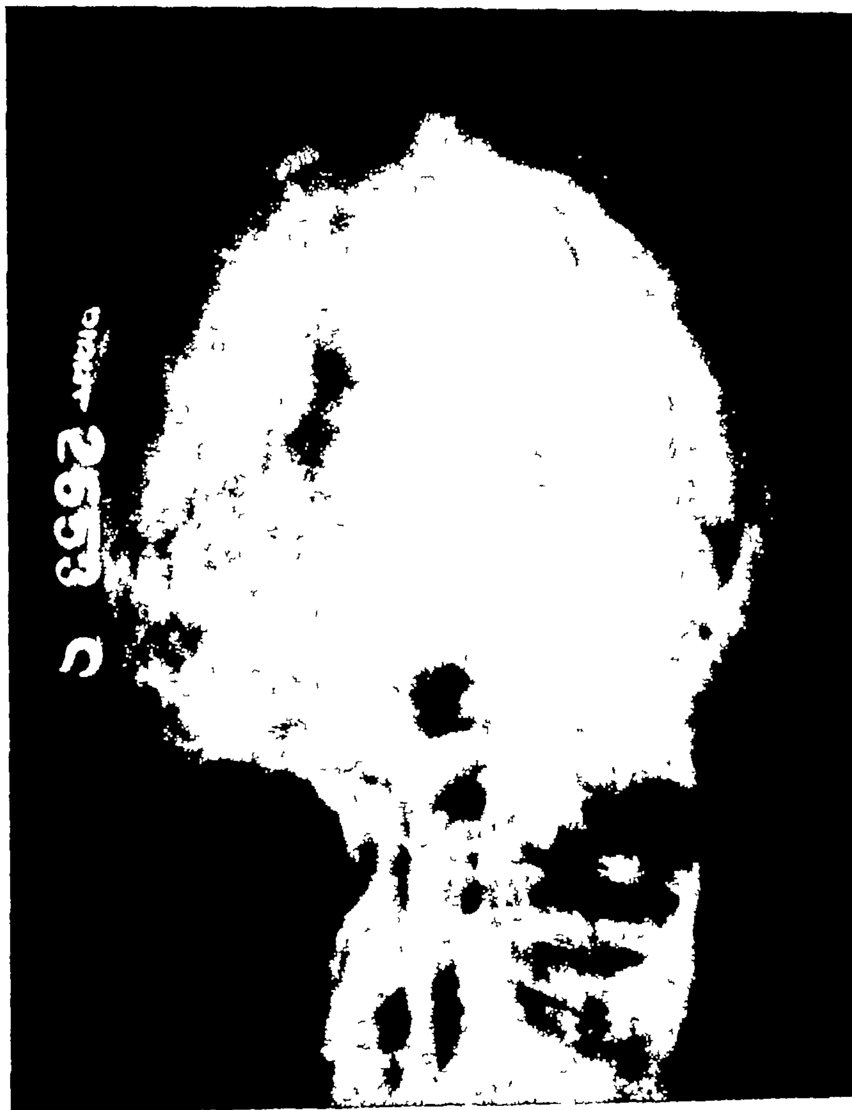


Fig 25 Ventriculogram showing displacement of the ventricular system to the left in right subdural hematoma

25) The technique of encephalography and ventriculography is described in Chapter XV.

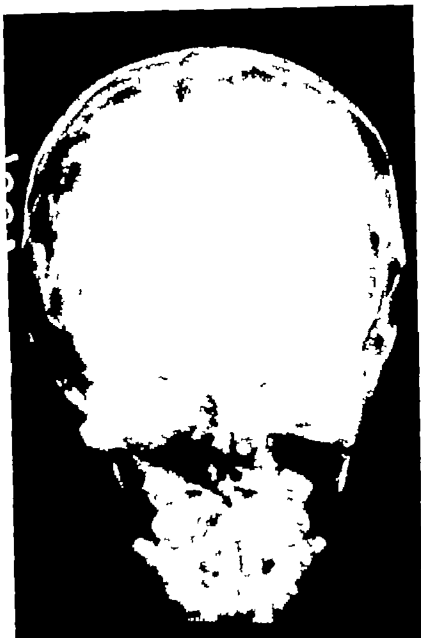


Fig. 28 Operative defect of the skull There is no evidence of regeneration of bone after four years.

HEALING OF SKULL FRACTURES

Reports of healing of fractures of the skull as demonstrated by periodic x-ray examinations taken months and years following the fractures are few Vance studied 52 cases In 36 of the patients there was no evidence of the fracture after two years In children, complete disappearance of a linear fracture may occur as early as four and one-half months after the injury

Operative defects of the skull and those resulting from the débridement of compound depressed skull fractures show *little or no evidence of regeneration of the bone even after* several years (Fig 26) For this reason one should always attempt to replace fragments of bone, wherever possible, during operations for depressed or compound depressed skull fractures

Chapter V

CLASSIFICATION OF HEAD INJURIES

A satisfactory classification of head injuries presents many difficulties from clinical, pathological and statistical view points The terms "concussion" and "skull fracture," though widely used by medical legal journalistic and lay individuals have done much to confuse and retard the understanding of head injuries "Concussion" becomes meaningless when applied alike as it often is, to the patient who has suffered a mild blow with temporary loss of consciousness and to the one who has had a severe brain injury with coma or delirium persisting for several days The term "skull fracture" is also often employed to designate any injury to the head even before there is evidence to show that there has really been a dissolution of continuity of the skull. Recently the terms "traumatic stupor" and "traumatic delirium" have been suggested to supplant the term "concussion" They merely describe the mental state of the patient, but disregard the severity of the injury the probable prognosis and the pathology A classification of head injuries based on etiology is of limited value While similar accidents may produce injuries of a similar or related type, they are just as likely to produce totally dissimilar injuries A classification on a physio-pathological basis is desirable but in most cases can indicate only an approximation of the alterations in the living patient

In actual practice the terms "head injury" or "craniocerebral trauma" have proved most useful These terms are inclusive—they indicate that the scalp cranium and intracranial

contents have been damaged, without focusing attention on nonessential details Often, it is impossible to determine the degree of injury until the patient has been observed for several hours The following classification is suggested

Mild head injury Loss of consciousness of not more than 10 minutes Minimal or no abnormal neurological signs, little or no shock, and no abnormal findings in the x-ray film.

Moderate head injury Period of unconsciousness lasting more than 10 minutes, positive neurological findings, positive x-ray findings and moderate shock The presence of any *one* of these is sufficient for a diagnosis of moderate head injury

Severe head injury Period of unconsciousness of an hour or more followed by delirium or clouding of sensorium lasting from several hours to several days, neurological abnormalities, x-ray or clinical evidence of fracture of the skull, moderate to severe shock

Extremely severe head injury Deep coma, severe shock, extensive neurological abnormalities, x-ray or clinical evidence of fracture of the skull In this group, death often occurs within the first twelve to twenty-four hours

The diagnosis of head injury should, then, be amplified as adequately as the facts at our command permit If laceration or hematoma of the scalp exists, or intracranial bleeding is demonstrated, the diagnosis should include a statement to this effect For example, a complete diagnosis would be Moderate head injury, with hematoma of left parietal region, fracture of left parietal region extending into base, probable contusion of left parietal lobe, and traumatic subarachnoid hemorrhage Such a diagnosis gives an adequate description of the entire picture and is most useful from both therapeutic and prognostic viewpoints.

INCIDENCE AND ETIOLOGY OF HEAD INJURIES

Head injuries have increased greatly in recent years This increase has been largely due to the widespread use of the automobile since hazardous industries are rigidly inspected and safeguards are mandatory

In 1936 there were 864 800 automobile accidents resulting in death and injury 36,800 were killed, and 967,840 injured In 1937 40 300 deaths and 1,220 000 injuries resulted from automobile accidents About 50 per cent of all persons killed in automobile accidents died of head injuries Many more received severe head injuries resulting in permanent disability Falls, assaults accidents at work at home, and at play swell the total of head injuries to a tremendous number

The following tabulation of the types of accidents resulting in head injuries is taken from a study of 444 cases observed in the Morrisania City Hospital, New York, during 1935 and 1936

TABLE I

Types of Accidents Resulting in Head Injuries

Type of Accident	*Number of Cases*	*Per Cent*
Pedestrian struck by automobile	124	28
Collision of automobiles	37	8
Simple fall	85	19
Fall from building or other high place	74	16
Assault	45	10
Struck by falling object	28	6
Motorcycle	9	2
Fall during convulsive seizure	3	0
Attempted suicide by leaping from roof	5	1
Bicycle	1	0
Struck by train	2	0
Not known	31	6

It is important to learn if possible the way in which th head injury occurred If the accident was of sufficient sever ity the injury must be considered severe until proved other

wise by careful observation, preferably in a hospital Head injuries are often considered "minor," and the patient is sent home after the suturing of a scalp laceration or a brief examination, only to develop serious symptoms a few hours or days later A case in point is the following

Case 1 A white male, 54 years old, a chef by occupation, was thrown to the pavement when hit by an automobile. He was dazed for an instant, but was soon able to get up by himself, and was permitted to go to his home by an ambulance surgeon who saw him soon after the accident An hour later he began to vomit and shortly thereafter he became confused and weakness developed in his left lower extremity A few hours later he became stuporous A physician who visited the patient at his home diagnosed "concussion of the brain" and prescribed rest in bed and an ice cap to the head The next morning the patient was deeply comatose and he was removed to the hospital

Examination revealed a patient who was deeply stuporous The pulse rate was 96, blood pressure 150/130 The pupils were equal and reacted to light. All extremities were spastic, the left more than the right The Babinski sign was present on both sides A diagnosis of right extradural hemorrhage was made and the patient operated upon immediately A large extradural clot resulting from a tear of the middle meningeal artery was found and removed The patient subsequently made a complete recovery

In every case of head injury in which there has been any disturbance in the state of consciousness, or in which the provoking accident was competent to produce a serious injury, the patient should be hospitalized, and carefully studied in order to rule out any damage to the brain, its vessels, or membranes

The initial disturbance in the state of consciousness may vary from a momentary period of confusion to deep coma lasting for many hours, days, or even weeks In mild head injuries the patient may be dazed and confused for a short period, then rapidly regain full awareness of his surroundings

ad injuries are complicated by other conditions — alcoholism epilepsy or metabolic disorders and it is difficult to determine which factor is playing the greatest rôle in the production of the altered state of consciousness Until proved otherwise the head injury must be charged with the major proportion of the symptoms and proper treatment and observation instituted.

Mild Head Injuries (Concussion)

There is no agreement concerning the cause of the initial loss of consciousness in mild head injuries The rapid restoration to normal within a short period of time would seem to preclude any structural change such as contusion edema petechial hemorrhages or increased intracranial pressure Some observers believe that an injury to the head results in a mechanical "shaking up" of the brain with a molecular disruption of the ganglion cells (commotio cerebri), but no convincing proof has been advanced to substantiate this theory The rapid recovery from unconsciousness suggests the operation of a quickly reversible reaction such as a temporary paralysis or depression of the vasomotor center with cerebral anemia or stasis The demonstration of vasomotor nerves in the cerebral vessels makes such a theory tenable

The prolonged coma of severe head injuries is due to actual anatomical changes in the brain—contusion hemorrhage and laceration In many patients who remain comatose for long periods the intracranial pressure as measured by spinal puncture and by observation of the optic discs remains within normal limits If a space containing lesion such as an epidural hemorrhage subdural hemorrhage or abscess develops the pressure may rise Zierold studied the spinal fluid pressure in 128 patients with head injuries who were unconscious for

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one hour or longer, in only 21 of them was the pressure 180 mm or more

In a carefully studied series of 444 patients with acute head injuries, spinal punctures were done in 71 in whom persistent headache, neck rigidity, or elevation of temperature was present The cerebrospinal fluid pressure was above 200 mm. of water in only 18

Shock associated with head injuries, unless accompanied by other serious injuries, is usually not severe In mild head injuries, unconsciousness lasts only a few minutes, and there are no marked changes in the pulse, blood pressure, respiration, or temperature As the patient returns to consciousness he may at first appear somewhat bewildered, but soon regains complete awareness of his surroundings, though he may have complete amnesia for the accident and the events immediately preceding it Headache may be present, and pain and tenderness experienced at the site of impact It is unusual to find any alteration in the neurological status in such cases. In alcoholic or senile individuals, a mild head injury may precipitate a marked alteration in the mental status the alcoholic addict may become wildly delirious, and most difficult to manage, the senile person who has suffered a mild head injury becomes confused, restless, irritable, and may have a muttering delirium

In most cases of mild head injury there is a tendency for the acute symptoms to clear up spontaneously If any untoward delay occurs in recovery it is evidence of damage to the brain, or of extracerebral hemorrhage

Moderate and Severe Head Injuries

In head injuries of greater magnitude than "mild" there is actual anatomical damage to the brain, and the disturbance in consciousness commonly lasts for an hour or more, de-

pending on the degree of injury, and on the general condition of the patient (age, influence of drugs or alcohol complicating injuries or disease) In these patients there is usually some evidence of shock the skin is moist and cold, respiration shallow pulse rapid, blood pressure low and the temperature at first subnormal. The neurological examination usually reveals abnormalities It is impossible to determine the gravity of a head injury from a single sign or symptom The state of consciousness however pulse rate, blood pressure temperature respiration, condition of the pupils neurological signs, and x ray examination of the skull all yield important diagnostic information

The disturbance of consciousness following a major head injury may be profound. Deep coma lasting for many hours is usually indicative of extensive contusion and laceration of the brain When this persists for several hours without any sign of motor activity the outlook is almost always hopeless In more favorable cases, stimulation of the patient produces some sign of motor activity, and he may become very restless when disturbed for examination or therapy Following a period of deep coma a stupor from which the patient can be aroused may persist for several hours or days In this state the patient can be made to answer questions and take nourishment The return of complete awareness is gradual and may take two to three weeks or longer In this interval confusion and delirium are frequent complications. The delirium may be mild and easily controlled, though often it is severe, and accompanied by marked restlessness excitement and impulsive acts

The pulse rate following an injury to the head must be viewed in correlation with other information Alone it is not of great value While the patient is still in shock the pulse may be rapid and irregular As recovery from shock occurs the

pulse becomes full and slower During convalescence, it is not unusual for the pulse rate to drop to sixty or less, without other symptoms. Nevertheless, every patient with a slow pulse deserves close observation and frequent eyeground examinations, especially if headache is present Slowing of the pulse with deepening stupor is frequently found in patients with extradural or subdural bleeding, even without other evidence of compression of the brain

In animal experiments (Cushing, Malone, and others), when the intracranial pressure is raised rapidly the pulse slows and the blood pressure rises as long as the medullary centers are able to compensate When compensation breaks the blood pressure falls, the pulse becomes rapid and irregular, and the respirations rapid Malone found that in animals compensation occurred as long as the pupils reacted to light When they failed to do so, medullary compensation to increased intracranial pressure was absent

Clinically, marked changes in blood pressure do not often occur following injuries to the head While the patient is in shock, the blood pressure is low. Progressive elevation of the blood pressure is seldom found except in some cases of middle meningeal hemorrhage, and even in these it is not a constant finding

The differences between the findings in experiments on animals, where the four stages of compensation to increased intracranial pressure can be regularly demonstrated, and in patients with brain trauma, do not invalidate the experimental data Following brain trauma the medullary centers are often depressed or damaged so that they are unable to respond, and it is rare to have marked and rapid elevation of intracranial pressure If the medullary centers are able to respond, and if the intracranial pressure rises rapidly we

find in patients the same changes that are regularly produced in the laboratory animals

Immediately following a head injury the temperature may be normal, and even subnormal, depending on the severity of the associated shock. As recovery from shock takes place the temperature returns to normal unless extensive cerebral damage is present In the absence of complicating factors elevation of temperature is a fairly reliable index of the degree of brain contusion and laceration Rapid elevation of the rectal temperature to 104° F., or higher is rarely present in patients who recover, though exceptions to this dictum are occasionally seen The febrile reaction seems to be a response to general brain injury and is of no localizing value although hemorrhage into the ventricles or around the medulla oblongata is usually accompanied by marked disturbances of temperature regulation

Alterations in respiratory rate and rhythm signify severe cerebral injury During the shock period respiration is shallow and weak. When conditions are favorable normal rate and rhythm is soon restored. Deep stertorous breathing accompanies compression of the brain due either to an extradural or subdural hemorrhage If compression is not relieved, irregular respiration of the Cheyne-Stokes type supervenes and may continue for several hours before death takes place When this stage begins, recovery is rare even after compression has been relieved

Information gained by an examination of the eyes is often difficult to interpret In mild head injuries pupillary and ocular abnormalities are ordinarily absent unless there has been direct trauma to the eye or orbit It is important to differentiate a traumatic mydriasis from the dilated pupil found in intracranial lesions This, as a rule presents no difficulties unless there is a coexisting severe intracranial injury When

the dilatation of the pupil is due to direct injury to the eye there will be some conjunctival hemorrhage and edema. It should be remembered, also, that a severe blow to the eye may produce a very slow pulse in individuals who have a sensitive oculocardiac reflex The combination of slow pulse and dilated pupil following an injury should make the observer wary, an extradural hemorrhage must be ruled out If the eye injury alone is responsible for the slow pulse and dilated pupil no other neurological abnormalities will be found

Pinpoint pupils following head injuries are often due to morphine given by the medical attendant who first saw the patient, although most ambulance surgeons now wisely refrain from administering morphine when an intracranial trauma is suspected (see page 76)

In moderate and severe head injuries nearly any type of pupillary disturbance may occur Certain changes have been quoted frequently by various authors as being of diagnostic significance, and some observers attach great importance to them, but changes in the pupils alone should never be the sole factor influencing any method of treatment It is true that a unilaterally dilated and fixed pupil is found more often on the side of the compressed hemisphere, but the opposite condition prevails often enough to warrant bilateral exploration when a suspected clot is not found on the side of the large, fixed pupil.

Bilaterally dilated, nonreacting pupils presage an unfavorable prognosis as a rule, although a rare exception may occur The condition of the pupils may change rapidly so that frequent observation and careful notes must be made in every case. While eyeground examinations should be made early and frequently, choked discs have rarely been noted earlier than the fourth or fifth day after a head injury.

Occasionally, swelling of the discs, in conjunction with

headache and a high spinal fluid pressure and without focal signs is noted during the end of the first week of a head injury This is probably due to a disturbance of cerebrospinal fluid absorption or secretion The cerebrospinal fluid balance can often be restored and regulated by spinal punctures Indeed one spinal puncture may be sufficient to re-establish a normal state of affairs

Alterations of reflexes and motor power are commonly found after head injuries With shock, all the tendon reflexes are greatly depressed or absent and the extremities flaccid As recovery from shock takes place the deep reflexes slowly return, so that at the end of twelve or twenty four hours they are normal or even hyperactive In severe head injuries especially in cases of basal skull fracture as recovery from shock occurs the extremities may become spastic and the reflexes hyperactive with abnormal plantar responses Persistent inequality of the reflexes is associated with a lesion in or near the motor area This may be accompanied by a marked weakness of the contralateral side As a rule the paralysis clears up though the reflex changes may persist for a long time When neurological abnormalities are found there has always been some contusion and perhaps laceration of the brain When the injury results from a direct blow by a blunt object the focal signs such as hemiplegia aphasia or hemianopsia may be more prominent than general signs of injury whereas with a diffuse blow such as is suffered from a fall from a height or an automobile accident the focal signs are often masked by the evidence of widespread damage to the brain

Convulsive seizures are not a very frequent complication during the first few days following a head injury They occurred during the first twenty four hours in only 6 of 444 patients with acute head injuries They may be focal (Jack

sonian) or generalized. When seizures occur they often tend to dominate the scene so that other important data may not be given proper weight Seizures occurring early (within the first few hours) are most often due to irritation of the motor cortex by subpial or intracortical hemorrhage or by a depressed bone fragment

Incontinence or marked restlessness, which persists after the patient has regained consciousness, is an indication of frontal lobe injury Glycosuria, in the absence of diabetes and before glucose has been given intravenously for therapeutic purposes, points to hypothalamic damage

Chapter VI

PRINCIPLES GUIDING THE TREATMENT OF HEAD INJURIES

The fundamental principles in the treatment of head injury include rest, quiet sufficient nourishment adequate fluid mineral and vitamin intake, attention to the bowels bladder and skin, combined with careful and skillful observation to detect changes requiring alteration in therapy Every patient must be treated as an individual and no rules can be formulated which are applicable to all cases In this, as in all good medical and surgical practice the treatment must be made to fit the patient, and the therapist must have in mind just what is expected of any proposed procedure

Mild head injuries require very little active therapy other than rest and quiet. After a period of observation of two or three days the patient may be permitted to go home but must be kept in bed for an additional week and then allowed out of bed only if he is symptom free With any head injury of greater magnitude than mild, a minimum of three weeks rest in bed is prescribed, and this may be prolonged if symptoms are present at the end of three weeks If possible the patient who has suffered a major head injury should be kept in a quiet, darkened room where his visitors can be restricted and where all disturbing influences can be eliminated The best "splint" for an injured brain is absolute rest—both physical and mental Outside stimuli can be best reduced in a quiet darkened room with careful and understanding nursing care

When a patient who has suffered a major head injury is first

brought to the emergency ward, there is a tendency to do too much. If shock is present it should be treated promptly, that is, as soon as a preliminary appraisal of the patient's condition has been made Fluids are given intravenously, combined with caffeine sodium benzoate If the patient is in severe shock an injection of 500 c c or more of 10 per cent glucose solution is advisable, otherwise 250 c c of 25 per cent glucose solution may be used ° The latter has a greater dehydrating effect, which may be desirable in some cases While the patient is in shock he should be kept flat in bed, warm and quiet If any lacerations are present they should be dressed with a sterile compression bandage. If other injuries are present they should be treated conservatively (unless there are urgent indications for surgical intervention). Fractures of the extremities should not be manipulated, or wounds sutured, or any other disturbing diagnostic or therapeutic procedures carried out It is never justifiable to move a patient or disturb him for x-ray examination while shock or extreme restlessness is present When the patient's condition has become stabilized roentgenograms of the skull may be made, wounds attended to, and tetanus antitoxin given if the nature of the injury requires it—that is, injuries such as open scalp wounds incurred in street accidents, or any contaminated wound Mishaps can be avoided if a history regarding allergy is obtained from the patient or a relative, and if sensitivity tests are carried out before the antitoxin is given

DEHYDRATION

˘ dehydration in the management of major
an unsettled problem In the minds of
ved hypertonic glucose solutions in-
some cases of acute head

injury marked benefit has resulted On the other hand many practitioners feel secure in treating a head injury routinely by dehydration and lumbar puncture However they may thus overlook a surgical emergency Theoretically hyper tonic solutions injected into a vein should reduce brain vol ume by abstracting fluids from the tissues, and there is abundant laboratory and clinical evidence that this takes place with a resultant reduction of intracranial pressure The secondary rise in intracranial pressure which occurs in experimental animals some hours after a single injection of hypertonic glucose has also been observed in traumatized in dividuals and indicates the need of a systematic regime when ever this form of therapy is employed Fay has combined the intravenous administration of 50 per cent glucose with rigid fluid restriction and magnesium sulphate by mouth We can not subscribe to the marked fluid restriction which Fay ad vises especially since the studies of Fremont Smith Merritt and Lennox have shown that in normal subjects with active diuresis the cerebrospinal fluid pressure was not influenced by the water intake Indeed drastic dehydration is often harmful and we believe definitely retards recovery Follow ing too enthusiastic dehydration fever delirium and restless ness may occur In such a patient a lumbar puncture may show a very low cerebrospinal fluid pressure in some in stances no fluid is obtained until the patient s head is ele vated Leriche described cases of hypotension of the cerebro-spinal fluid following trauma, and we also have seen this syndrome which clears up rapidly when hypotonic fluids are given liberally by mouth or intravenously in comatose patients

We have adopted the following policy regarding the use of hypertonic solutions in head injuries

1 Hypertonic glucose is given to every patient who is

brought to the emergency ward, there is a tendency to do too much If shock is present it should be treated promptly, that is, as soon as a preliminary appraisal of the patient's condition has been made Fluids are given intravenously, combined with caffeine sodium benzoate If the patient is in severe shock an injection of 500 c c or more of 10 per cent glucose solution is advisable, otherwise 250 c c of 25 per cent glucose solution may be used * The latter has a greater dehydrating effect, which may be desirable in some cases While the patient is in shock he should be kept flat in bed, warm and quiet If any lacerations are present they should be dressed with a sterile compression bandage If other injuries are present they should be treated conservatively (unless there are urgent indications for surgical intervention) Fractures of the extremities should not be manipulated, or wounds sutured, or any other disturbing diagnostic or therapeutic procedures carried out It is never justifiable to move a patient or disturb him for x-ray examination while shock or extreme restlessness is present When the patient's condition has become stabilized roentgenograms of the skull may be made, wounds attended to, and tetanus antitoxin given if the nature of the injury requires it—that is, injuries such as open scalp wounds incurred in street accidents, or any contaminated wound Mishaps can be avoided if a history regarding allergy is obtained from the patient or a relative, and if sensitivity tests are carried out before the antitoxin is given

DEHYDRATION

The question of dehydration in the management of major head injuries is still an unsettled problem In the minds of those who have employed hypertonic glucose solutions intravenously there is no doubt that in some cases of acute head

* Quantities are for the average adult

injury marked benefit has resulted. On the other hand, many practitioners feel secure in treating a head injury routinely by dehydration and lumbar puncture. However, they may thus overlook a surgical emergency. Theoretically, hypertonic solutions injected into a vein should reduce brain volume by abstracting fluids from the tissues, and there is abundant laboratory and clinical evidence that this takes place with a resultant reduction of intracranial pressure. The secondary rise in intracranial pressure which occurs in experimental animals some hours after a single injection of hypertonic glucose has also been observed in traumatized individuals and indicates the need of a systematic regime whenever this form of therapy is employed. Fay has combined the intravenous administration of 50 per cent glucose with rigid fluid restriction and magnesium sulphate by mouth. We cannot subscribe to the marked fluid restriction which Fay advises, especially since the studies of Fremont Smith, Merritt and Lennox have shown that in normal subjects with active diuresis the cerebrospinal fluid pressure was not influenced by the water intake. Indeed, drastic dehydration is often harmful, and we believe definitely retards recovery. Following too enthusiastic dehydration, fever, delirium and restlessness may occur. In such a patient a lumbar puncture may show a very low cerebrospinal fluid pressure; in some instances no fluid is obtained until the patient's head is elevated. Leriche described cases of hypotension of the cerebrospinal fluid following trauma, and we also have seen this syndrome which clears up rapidly when hypotonic fluids are given liberally by mouth or intravenously in comatose patients.

We have adopted the following policy regarding the use of hypertonic solutions in head injuries:

1. Hypertonic glucose is given to every patient who is

admitted to the hospital in coma or in shock When shock is severe, 500 c c. of 10 per cent glucose are given, otherwise 250 c.c of 25 per cent glucose Hypertonic solutions of glucose given by vein increase the circulating fluids and thus aid in combating shock In addition, the glucose affords a readily available nutriment to the organism

2 When focal or general cerebral edema is suspected the reduction of brain bulk can be rapidly accomplished by hypertonic solutions We have found them especially beneficial in the treatment of focal seizures in children, where there is no clinical or x-ray evidence of a depressed skull fracture or middle meningeal hemorrhage

3 When an extradural hemorrhage or subdural hematoma is suspected, hypertonic solutions are not given, but trephine exploration is done at once. In these cases hypertonic solutions may be dangerous, since the reduction of brain volume may accelerate the bleeding or permit new bleeding when it has already stopped

The choice of the solution used depends on the experience of the therapist Hypertonic solutions of sodium chloride were first employed However, their administration is often followed by severe respiratory and cardiac disturbances and therefore glucose is now most often used. Recently, sucrose has enjoyed popularity, and is advantageous since it is less frequently followed by a secondary increase in intracranial pressure Other complex sugars such as sorbitol, have been advocated recently Severe chills have occurred after their use, however, and they have not been employed long enough to determine their place in the treatment of increased intracranial pressure following head injuries Solutions of glucose have the advantage of supplying a readily available source of energy, and when properly prepared and administered, untoward reactions can practically be eliminated.

The concentration and amount of hypertonic solution in jected intravenously, depends on the degree of reduction in intracranial pressure which is desired, and on the presence and severity of shock. Glucose is ordinarily given in concen trations of 5 10 25 or 50 per cent When shock is severe 500 c c. of 10 per cent glucose are administered to the aver age adult When a rapid reduction in intracranial pressure is desired 250 c c. of a 25 per cent solution or 100 c c of a 50 per cent solution is adequate Fifty per cent glucose has the dis advantage of occasionally causing thrombosis of veins Su crose is usually administered in a 50 per cent solution, 100 c.c. at a time As soon as the patient's condition permits dehydra tion if desired, can be maintained by 50 per cent magnesium sulphate given by mouth or as retention enemata. Rectal ad ministration of 50 per cent magnesium sulphate solutions is a convenient method of administering hypertonic solutions For adults 150 to 180 c. c. of the solution warmed to body tem perature should be given slowly by means of a small catheter and funnel To obtain full effect the solution must be retained for twenty to thirty minutes The magnesium sulphate should be continued until the patient has had several watery stools If there is the least suspicion of a coexisting abdominal in jury magnesium sulphate solution is of course not given

POSITION OF THE PATIENT

No general rule can be stated which is applicable to all cases If the patient is in shock he should be kept flat or the foot of the bed elevated but slightly When the blood pressure has returned to normal the head of the bed may be raised slightly unless there is a tendency for fluid to collect in the lungs and respiratory passages In such cases elevation of the foot of the bed with the patient lying on his side will ma terially aid in the removal of the fluid In general most

patients are more comfortable with the head rest slightly raised since in this position intracranial pressure is lower than in the prone position, and venous stasis is relieved.

SEDATION

Narcotics are dangerous because of their depressing effect on the medullary centers, and also because they interfere with observation of the patient. In addition, morphine constricts the pupils, limiting pupillary reactions and fundus examinations. There is some evidence, too, that morphine causes a rise in intracranial pressure. Inasmuch as the state of consciousness is a most valuable index of the patient's progress, the use of depressants is usually contraindicated. When drugs are required for restlessness, the administration of sodium phenobarbital by hypodermic injection, paraldehyde, and sodium bromide combined with chloral, by mouth or rectum, has proved of value.

The sedative chosen and its dosage depends on the degree of restlessness and the reaction in the individual case. It is our practice to begin with the administration of 2 Gm. (30 grains) of sodium bromide combined with 0.5 Gm. (7½ grains) of chloral hydrate. This may be repeated every four or six hours as indicated.

Phenobarbital and sodium phenobarbital are used to allay moderate restlessness. For oral use, 0.1 to 0.2 Gm. (1½ to 3 grains) of phenobarbital is required. If restlessness persists, paraldehyde, 8 to 12 c.c. (2 to 3 drams) may prove efficacious. The disagreeable taste of paraldehyde may be disguised by dispensing it combined with orange juice and ice cubes.

When it is impossible to administer sedatives by mouth, rectal or hypodermic medication must be resorted to. Sodium bromide and chloral hydrate as well as paraldehyde can be given in the above dosages in small enemata. Such adminis-

tration should be preceded by cleansing the rectum with warm water

For hypodermic use, sodium phenobarbital 0 13 to 0 3 Gm (2 to 5 grains) is the drug of choice in head injuries

DIET

The diet for a patient with a head injury depends more or less on the patient's appetite It should be light but the caloric, mineral and vitamin content must be adequate especially when the patient remains in bed for many weeks At first, fluids and easily digestible food such as broths custards and soft boiled or poached eggs are given As the patient recovers the diet should be increased as tolerated so that when he is up and about it is practically normal If coma persists for several days or weeks, feeding and the maintenance of a normal metabolic balance may become a major problem Fluids sodium chloride and glucose may be given intravenously However when this is done the fluid and sodium chloride intake must be carefully calculated to maintain a proper electrolytic balance The rules formulated by Coller provide the best guide While Coller was especially interested in the postoperative care of surgical patients, the principles of his treatment apply equally well to patients with head injuries who require parenteral fluids for a prolonged period

The following method is used to calculate the fluid requirements. Normally 800 to 1500 c c. of water a day are needed for vaporization (loss through lungs and skin) with profuse diaphoresis more fluid is required The kidneys need about 1500 c c of water a day for the elimination of an average of 35 Gm of solid waste material approximately 200 c c. of water is lost with the feces This adds up to 2500 to 3700 c c a day To this must be added any abnormal loss of fluid by

vomiting, diarrhea or profuse sweating Dehydration will result if the fluid intake does not equal the fluid loss. While the average patient may tolerate some degree of dehydration, those in poor condition or those who remain in coma for many days will show signs of fluid deprivation A dry tongue, loss of turgor of the skin and sunken eyes, combined with concentrated urine containing a trace of albumin and casts, are indications of such deprivation In this condition, the patient has usually lost about 6 per cent of his body weight in fluids For an average individual of 60 Kg this amounts to a loss of 3600 c c A daily urine output of 1500 c c of a specific gravity of 1 015 or less, normally indicates that the fluid intake has been adequate.

In determining the sodium chloride requirements the following facts are important The usual salt loss in feces and sweat is about 0 5 Gm a day The remainder of the ingested salt, amounting to from 6 to 12 Gm a day, is excreted in the urine When fluids are given parenterally excess salt administration must be avoided If the daily fluid requirements are given in the form of 3000 c c of 5 per cent glucose in saline the patient receives 25 5 Gm. of sodium chloride This results in a retention of salt and water, causing edema, and is especially liable to occur in patients who have been ill or in coma for a long time and whose serum proteins are low

On the other hand, an inadequate salt intake causes a loss of body electrolytes, the symptoms of which are lassitude, asthenia, dulling of the sense of taste, nausea, vomiting, drowsiness and later, coma There is usually an associated dehydration The degree of salt deprivation can be estimated by a plasma chloride determination. To bring the blood chlorides to normal (580 mg per 100 c c) 0.5 Gm. of sodium chloride per kilogram of body weight should be given for each 100 mg that the plasma chloride level needs to be ele-

vated The following case illustrates the harm which may arise from an inadequate sodium chloride intake

Case 2. The patient, a white male 50 years old was injured in an automobile collision. He became unconscious immediately and remained so for several days He was given hypertonic glucose in distilled water by vein two or three times each day No saline solution was administered. When seen by one of the authors several days after the accident he was in profound coma. His skin was dry and inelastic, his tongue dry and furrowed, and the temperature was 103 6 F It was evident that the patient was suffering more from the treatment which he had received than from his injury Normal saline solution was given by continuous intravenous drip Twelve hours later the patient s temperature had fallen to 101 F and he was no longer in coma, though still stuporous A Levine tube was introduced and a high caloric balanced liquid diet given by this method. At the end of two days the patient had improved sufficiently to take fluids by mouth and he eventually recovered completely

From our knowledge of the water salt caloric and vitamin requirements an adequate diet can be provided even for patients who remain comatose for several days or who for other reasons are unable to take food by mouth The total sodium chloride should average 5 to 6 Gm a day the total fluids about 2500 c c and the caloric intake should be from 1500 to 1800 calories This can be made up of sweetened fruit juices egg nogs milk cream and other fluids of high caloric value Any vitamin deficiency due to the restricted diet can be made up by the administration of synthetic preparations

LUMBAR PUNCTURE

The place of lumbar puncture in the management of head injuries has provoked much controversy Some physicians believe lumbar puncture dangerous and never employ it in

acute head injuries Other equally competent observers use lumbar puncture in every case and believe it beneficial and safe Lumbar puncture as a diagnostic procedure is rarely necessary in head injuries, though it may be of value when a definite history of trauma cannot be obtained, and when there is no external evidence of injury to the head In most cases of moderate and severe head injuries (about 90 per cent) a bloody fluid is obtained by lumbar puncture It is hazardous to attempt to estimate the severity of the injury by the amount of blood in the fluid Nevertheless, large amounts of blood are usually indicative of extensive cerebral contusion and laceration with tearing of cortical vessels Due to the frequently associated contusion of the brain, bloody fluid is found in many cases of extradural hemorrhage

The cerebrospinal fluid pressure is not often elevated immediately following a head injury Errors are frequently made, due to restlessness of the patient When a block is present, such as might result, for example, from the lodgment of a blood clot in the aqueduct, or around the fourth ventricle, the pressure determined by lumbar puncture does not correspond to the intracranial pressure How often such a block occurs following head injuries is not known since in postmortem studies very little attention has been given to this until recently, when Moritz and Wortman reported four cases of acquired internal hydrocephalus in which there were histories of severe head injury antedating the disease The hydrocephalus in each case was due to a chronic obstructive leptomeningitis in the region of the roof of the fourth ventricle

The proponents of repeated lumbar puncture following head injuries believe that the intracranial pressure is thus maintained at a safe level, and that the removal of blood from the subarachnoid space hastens recovery and diminishes the

severity of the post-traumatic symptoms On the other hand, there are those who decry the use of lumbar puncture and consider it dangerous because of the possibility of herniation of the medulla oblongata through the foramen magnum following the sudden release of pressure The possibility of starting an arrested hemorrhage, due to changes in the intracranial structures caused by the removal of fluid from the ventricles and subarachnoid space by lumbar puncture, must also be considered. We have adopted the following policy regarding lumbar puncture

Lumbar puncture must not be done if shock or extreme restlessness is present, and is rarely indicated early in the course of a head injury unless necessary for diagnostic purposes During the first few hours following a head injury, the body attempts to restore the balance between intracranial, arterial venous and cerebrospinal fluid pressures The withdrawal of cerebrospinal fluid by lumbar puncture might possibly interfere with this. If an extradural hemorrhage exists the removal of cerebrospinal fluid from the ventricles and subarachnoid cisterns by lumbar puncture would afford a larger space in which the clot could form and fresh bleeding might occur In compound skull fractures and in fractures of the base with cerebrospinal rhinorrhea or otorrhea, early spinal puncture is contraindicated since it might tend to favor the passage of potentially infected material into the intracranial cavity

Lumbar puncture often greatly disturbs an uncooperative patient so that more harm than benefit results In very restless and uncooperative patients the hazard of breaking a needle in the spine must also be considered. If severe headache nuchal rigidity, or fever are present, after the condition of the patient has become stabilized spinal puncture may be of benefit The pressure should be measured and the

fluid removed slowly with a needle of small caliber (19 or 20 gauge), reducing the pressure to one-half of its initial reading. If the patient improves subjectively or objectively, puncture may be repeated every twelve or twenty-four hours, depending upon the symptoms. Spinal puncture is of greatest benefit to patients who develop severe headaches on the third or fourth day post-trauma, and in whom the spinal fluid pressure is elevated at that time. The removal of blood from the ventricles and subarachnoid spaces by lumbar puncture, on theoretical grounds at least, should prove beneficial. That blood is an irritant in the cerebrospinal fluid pathways was demonstrated by the work of Bagley. Autopsies on individuals dying some time after head injuries have shown thickening of the leptomeninges with hemosiderin pigmentation of the scar tissue (Moritz and Wortman). Parker and Lehman showed that in the experimental laceration of the brain in the dog, the cerebrospinal pressure varied directly with the amount of blood in the subarachnoid space, and we have frequently observed clinical improvement following the removal of bloody and xanthochromic cerebrospinal fluid by a series of lumbar punctures. It is difficult to reconcile these observations with the carefully controlled experiments of Sprong who showed that the rate of disappearance of blood from the cerebrospinal fluid is not appreciably influenced by repeated lumbar punctures.

SUBTEMPORAL DECOMPRESSION

Subtemporal decompression is now rarely advocated for the relief of intracranial pressure following head injuries. Experience has shown that unless a definite space-containing lesion, such as an extradural clot, or a subdural hemorrhage or collection of fluid, is disclosed, operations during the first twenty-four hours after a head injury, for the relief of pres-

sure, are usually of no benefit Nevertheless, there are th
who advocate subtemporal decompression For examp
Dandy believes that about 10 per cent of severe head injur
require subtemporal decompression for the relief of incre
ing intracranial pressure He cautions that it should not
done until at least six hours have elapsed from the time
injury Naffziger states that the conditions which jus
operation are more apt to be apparent after twelve or twer
four hours When a subtemporal decompression is do
Naffziger advises that the subdural space be drained
forty-eight hours

The studies of Mock Morrow and Shannon seem to c
demn the employment of subtemporal decompression Ho
ever we cannot subscribe to their dictum that "a skull fr
ture case operated upon immediately for any cause seld
lives" We have not employed subtemporal decompress
during the first twenty-four hours for the relief of increa
intracranial pressure resulting from head injuries for
following reasons

1 Increased pressure per se is not the most import
problem following severe brain trauma Most of the sy
toms are due to actual structural changes such as contus
laceration and hemorrhage These are not influenced by s
temporal decompression

2. The added trauma of operative procedure is often s
cient to weigh the balance against recovery when the
tient s fate is uncertain

3. Subtemporal decompression alone merely increases
cranial capacity Within a short time equilibrium betw
the general circulation and intracranial fluid contents is
established and the decompression becomes valueless

Subtemporal decompression is justified in cases in wh
after a lapse of twenty four or forty-eight hours or m

coma deepens in spite of dehydration and lumbar punctures Most of these patients die even though decompression is done, since the degree of cerebral damage is usually so great as to preclude recovery

In our experience, patients who have improved following subtemporal decompression were operated upon several days after their injury when the development of focal signs or increasing coma made it imperative to exclude a subdural or extradural clot Improvement followed negative explorations in a number of instances, and the subtemporal decompression was credited with the favorable outcome The following case is an example of this

Case 3 A white female, aged 32 years, a housewife, was injured in an automobile collision She was immediately rendered unconscious and was taken to the hospital, regaining consciousness after thirty minutes At that time a slight weakness of the left lower extremity was noted X-ray examination of the skull revealed a 4 cm horizontal linear fracture in the squamous portion of the right temporal bone, traversing the channel of the middle meningeal artery Lumbar puncture showed slightly bloody fluid, said to have been under increased pressure The patient gradually became more stuporous and slowly developed a definite left hemiplegia.

This patient came under our observation five days after the accident Examination at this time disclosed a deeply stuporous obese female She had a left hemiplegia There was moderate nuchal rigidity. The pupils were equal and reacted promptly to light The fundi showed early papilledema The pulse rate was 50 A right subtemporal decompression was decided upon No clot was found The dura mater was extremely tense and when opened the brain bulged through the dural decompression and did not pulsate The procedure was carried out under local anesthesia and when the dura mater was opened the patient regained consciousness on the table During the following two weeks the left hemiplegia rapidly cleared up. The patient has since remained well

Chapter VII

INJURIES OF THE SCALP AND SKULL

LACERATIONS OF THE SCALP

In most head injuries the scalp and skull are more or less traumatized Bruises and hematomas of the scalp require no special treatment other than cleansing In the presence of active and severe hemorrhage a scalp wound, of course, requires immediate attention In most cases however it is wise to delay débridement until an appraisal of the patient s condition is made Shock, for example needs immediate treatment, and when present a temporary antiseptic compression bandage may be applied It is better to obtain roentgenograms of the skull before lacerations are débrided and sutured, since occasionally unsuspected linear or depressed fractures are revealed at a distance from the scalp wound.

The first step in the repair of a scalp laceration is proper preparation of the patient. The hair is clipped and shaved to include a space at least two inches around the entire area An electric hair clipper greatly facilitates this procedure and is a useful and valuable addition to the equipment of every emergency surgery The next step is thorough cleansing of the entire area with green soap and warm water followed by alcohol and ether Since the scalp is thick and glandular and it is almost impossible to render it bacteria free painting with an antiseptic, such as a dilute tincture of iodine is advisable Next, a local anesthetic such as 1 per cent novocaine solution is injected at a distance of about one inch around the entire laceration With a scalpel the edges of the wound

are then excised en bloc down to the bone Hemostasis is obtained by finger compression (Fig. 77). Irrigation with saline or Dakin's solution is used The galeal edges are then



FIG 27 Almost complete avulsion of the scalp The wound was debrided according to the method described in the text, and healed by first intention The photograph was taken on the ninth postoperative day

approximated with interrupted sutures of fine black silk tied with three knots and cut as close to the third knot as possible These sutures remain buried and cause no trouble, if gentleness is used in handling tissues and all devitalized material removed Silk is used for the skin (Fig 27) Drainage is not employed. In many cases where a rapid closure of an

extensive scalp laceration is desirable because of the age or condition of the patient closing the wound with through and through silk sutures after preparation and débridement may serve the patient's welfare better than will a layer of buried silk sutures in the galea. The through and through sutures are loosely placed one-half to one inch apart, so that serum and blood which may collect can escape between the sutures

Neglected or improperly treated scalp wounds frequently become infected and the infection tends to spread in the loose areolar tissue between the galea and pericranium Such infected wounds are best treated by shaving the hair from a wide area and applying warm wet dressings The firmness of the scalp usually prevents gaping so that fairly good approximation can be obtained without the use of sutures If fluctuation occurs in areas remote from the wound free incisions may be made in these areas to obtain drainage

COMPOUND SKULL FRACTURES

A fissure fracture of the skull under a scalp laceration does not alter the treatment from that just described Every effort must be made to obtain primary healing The laceration is thoroughly debrided and closed in layers without drainage just as soon as the condition of the patient permits Dirt hair or other foreign material adherent to the fissure should be removed with a curette Unless symptoms warrant no attempt should be made to separate the edges of the fracture If exploration of the dura mater and brain is indicated it is best done by making a trephine opening to one side of the fracture

A lesson learned during the World War, and relearned many times in civil practice is this In the treatment of com

pound skull fractures thorough débridement and careful closure in layers with fine silk, without drainage, yields the best results

DEPRESSED SKULL FRACTURE

From the point of view of treatment, simple depressed skull fractures fall into three groups

1 Those which require early or immediate elevation In this group are included the depressed skull fractures which are responsible for serious symptoms, especially depressions over the motor area which produce Jacksonian motor convulsions or focal paralyses If a series of Jacksonian convulsions corresponding to the involved motor area occur, and cannot be controlled by sedatives, the depressed fragment of the bone should be elevated as soon as possible This is illustrated in the following case

Case 4 A white fireman, aged 52 years, was struck on the head with a hammer by a fellow employee He was stunned and taken to the hospital where he began to have frequent Jacksonian motor convulsions beginning in the fingers of the left hand and spreading to involve the entire left upper extremity When examined the patient was semistuporous The left upper extremity was powerless, though as far as one could tell painful stimuli were perceived in this extremity X-ray films of the skull (Fig 28) revealed a depression in the right parietal region, corresponding to the arm center At operation a fragment of bone about the size of a half a dollar was found depressed from three-eighths to one-half an inch The depressed fragment was removed (Fig 29) exposing a small tear in the dura mater A bleeding cortical vein was controlled with the electrocautery The wound was closed with fine silk sutures without drainage The patient made a rapid recovery He had no seizures subsequently, but there was some residual weakness and spasticity of the left upper extremity

If focal paralyses develop corresponding to the motor area

involved elevation of the depressed fragment is urgently indicated

2 This group includes cases of depressed skull fractures



FIG. 28. Case 4 Depressed fracture of the skull caused by a blow from a hammer

in which all the symptoms do not point directly to the involved area but many are due to general damage to the brain In this group it is wisest to delay elevation of the depression until the patient s condition has improved, and until the intracranial pressure has become stabilized within normal limits To elevate a depressed fracture in the presence of high intracranial pressure is to invite needless trouble and possible disaster Bleeding is greater and satisfactory closure of the wound difficult when the intracranial pressure

types of cranial surgery The surgeon should aim to le
the patient with as little defect in the skull as possible I
sating defects are a source of worry and potential dange
the patient. If the depression is large, the best incisior
horseshoe-shaped, with the depression in the center The
pressed fragment can then be raised by engaging an eleva
under an edge, and carefully prying it into place. Extre
caution must be employed to avoid injury to the underly
dura mater and brain Often it is necessary to make a treph
opening at one side of the depression so that an instrum
can be passed through it along the under side of the
pressed fragments Depressed fragments are freque
firmly wedged together and cannot be easily freed with
further damage to the underlying dura mater and brain
these cases by nibbling away a narrow margin of the b
at the edges of the cranial defect the impacted fragm
can be easily elevated If the dura mater is found intac
is wisest to leave it so, unless exploration of the brain is
dicated The dura mater may be incised because of neuro
ical signs or symptoms, or if inspection reveals a clot
collection of blood underneath When the dura mater
been torn it should be carefully closed with interrupted
sutures The defect in the skull is then repaired by fitting
larger fragments into place They can often be firmly wed
together by placing their edges in the diploic space betw
the two tables of the calvarium

If the dural edges have been extensively torn so that t
cannot be approximated, a transplant obtained from the t
poral fascia or the fascia lata can be used. The use of for
material such as prepared ox fascia lata is not advisabl
an early primary operation Likewise foreign material
repair skull defects are not advisable at the primary re

is high If the scalp over a depression is badly contused so that primary healing would be interfered with, it is wisest to wait until the condition of the scalp is no longer a barrier



Fig 29 Case 4 X-ray of the skull taken after débridement of the compound depressed fracture

to primary union Depressions in or near the motor area, even if symptomless, should be elevated when the condition of the patient allows

3 This group is comprised of cases of simple depressed fractures which need never be elevated Included are those patients in whom the depression is slight, or feeble or aged patients with a symptomless depression

The technique for the elevation of a simple depressed skull fracture does not differ from that employed in other

types of cranial surgery The surgeon should aim to leave the patient with as little defect in the skull as possible Pulsating defects are a source of worry and potential danger to the patient If the depression is large the best incision is horseshoe-shaped, with the depression in the center The depressed fragment can then be raised by engaging an elevator under an edge and carefully prying it into place Extreme caution must be employed to avoid injury to the underlying dura mater and brain Often it is necessary to make a trephine opening at one side of the depression so that an instrument can be passed through it along the under side of the depressed fragments Depressed fragments are frequently firmly wedged together and cannot be easily freed without further damage to the underlying dura mater and brain In these cases, by nibbling away a narrow margin of the bone at the edges of the cranial defect, the impacted fragments can be easily elevated If the dura mater is found intact it is wisest to leave it so, unless exploration of the brain is indicated The dura mater may be incised because of neurological signs or symptoms, or if inspection reveals a clot or a collection of blood underneath When the dura mater has been torn it should be carefully closed with interrupted silk sutures The defect in the skull is then repaired by fitting the larger fragments into place They can often be firmly wedged together by placing their edges in the diploic space between the two tables of the calvarium

If the dural edges have been extensively torn so that they cannot be approximated, a transplant obtained from the temporal fascia or the fascia lata can be used The use of foreign material such as prepared ox fascia lata is not advisable at an early primary operation Likewise foreign materials to repair skull defects are not advisable at the primary repair

of a depressed skull fracture, since such foreign bodies may be irritating to the tissues and thus lessen resistance to possible infection

COMPOUND DEPRESSED SKULL FRACTURES

A compound depressed skull fracture of any part of the skull demands early repair if intracranial infection is to be prevented As soon as the patient's condition permits, and within the first twenty-four hours if possible, thorough débridement and careful closure should be done The extent of the procedure required depends on the nature of the injury The following guiding principles apply to all compound depressed skull fractures

1 Local anesthesia whenever possible Avertin anesthesia for children Inhalation anesthetics, which tend to produce straining, struggling, or coughing, are contraindicated

2 Careful and thorough preparation of the scalp Usually clipping and shaving of the entire scalp, then cleansing with green soap, water, alcohol and ether

3 Trimming away with a sharp knife the contused scalp edges, temporal muscle if involved, and periosteum

4. Constant Dakin's or saline solution irrigation to remove dirt and other foreign materials

5 Elevation of the depressed fragments without injury to the dura mater or brain

6 If the dura mater is intact it must not be opened unless neurological signs or symptoms demand, or unless inspection reveals a subdural collection of blood or clot

7 If the dura mater has been torn and its edges contaminated or devitalized, they must be trimmed

8 Contused, macerated, and contaminated brain tissue must be removed by irrigation and suction with careful at-

tention to hemostasis Foreign bodies and fragments of bone are carefully removed

9 The wound is then closed in layers with interrupted fine silk sutures without drainage If the dural edges cannot be approximated a transplant of temporal fascia or fascia lata is used Large fragments of bone may be used to fill in the skull defect if they can be anchored in place

DEPRESSED SKULL FRACTURES IN THE NEWBORN

The general topic of cerebral birth injury is a volume in itself and has been adequately discussed in recent monographs (Ford Crothers Rydberg Ehrenfest) However injuries to the cranial bones in the newborn with minimal cerebral damage have not been stressed. Depressed skull fractures are usually due to improper application of forceps during delivery or pressure of the head against the pelvic bones during labor The skin covering the depression is rarely lacerated Many depressed skull fractures in the newborn are not productive of symptoms and spring back into place spontaneously after several days If the depression is marked or signs of cerebral compression or irritation occur then immediate elevation of the depressed area is indicated Focal seizures often subside as soon as a depressed fragment over the motor area is returned to its normal position

The technique of elevation is similar to that employed for other simple depressed skull fractures Operations in the newborn can be safely performed if loss of body heat and fluids is guarded against It is our practice to place the infant on a heated table This can be improvised by using an instrument tray under which one or two hot water bottles are placed Fluids are given subcutaneously about an hour before operation which is done under local anesthesia supplemented if necessary by a few drops of ether

REPAIR OF OLD SKULL DEFECTS (CRANIOPLASTY)

Skull defects resulting from compound skull fractures or cranial operations are sometimes a source of worry to the patient and are to some extent hazardous In addition, pulsating defects in the frontal region demand repair for cosmetic and social reasons Whether or not the cranial defect itself is productive of symptoms is a disputed point Nevertheless, dizziness on bending over, headache and rapid fatigability often clear up after repair of the skull defect Many patients suffer a psychological handicap a constant feeling of insecurity may accompany the knowledge that the calvarium is not intact Persons with visible skull defects may find it difficult or impossible to obtain employment In any of the above circumstances, cranioplasty is indicated, provided the wound has been thoroughly healed for at least six months and there is no evidence of infection in the skull or brain

Various substances have been used to repair cranial defects Silver plates, celluloid, and animal bone, are only a few For small defects it is preferable to use the patient's own outer table The scalp is prepared as for any other cranial operation A horseshoe-shaped flap is made, including the defect and an adjacent area of skull slightly larger than the defect The periosteum is incised close to the defect, and excess scar tissue is removed Care is taken to avoid injury to the dura mater. The margins of the defect are exposed with a periosteal elevator and freshened with a rongeur Adherent dura mater is freed from the under surface of the bone A piece of cellophane, cut to fit the defect, is then placed on the periosteum of the adjacent intact outer table With a scalpel an incision about one-fourth of an inch larger than the cellophane pattern is made in the periosteum The

edges of the periosteum are rolled over and an outline, slightly larger than the size of the pattern, is scratched around the bone with a sharp pointed instrument. With a mallet and chisel the outer table is cut through all around the pattern and then separated from the inner table Care should be used to prevent perforating the inner table The graft with the attached periosteum is transferred to the defect, and trimmed with a rongeur to secure a close fit It is then anchored by suturing the periosteum of the graft to the periosteum surrounding the defect with fine black silk For large defects, bone obtained from other individuals or at autopsy may be used A piece of bone larger than the defect to be repaired is obtained and its periosteum stripped The bone is boiled with the instruments The defect is exposed and prepared as already described From a cellophane pattern the bone graft is cut to fit snugly and then anchored into place by means of silver or stainless steel wire or black silk passed through drill holes in the skull and the bone graft The scalp flap is sutured into place in layers with fine black silk usually without drainage If a drain is used, it is removed within twenty four hours An adequate gauze roll dressing covered with a protective crinoline dressing is applied

The results of this operation on the whole have been good With careful technique it is rarely necessary to remove a bone graft because of non healing of the wound Roentgenograms taken many months later may show some decalcification of the graft but as absorption occurs there is replacement with tough fibrous tissue and the old defect remains firm without deformity or loss of protective function

In the following patient cranioplasty gave an excellent functional and cosmetic result

Case 5 A schoolboy 11 years old suffered a compound skull fracture Debridement was done leaving a large defect in the

left frontal region (Fig 30) The boy returned to school The pulsating defect kept him from playing with other boys or engaging in any sports After two years there was no evidence of

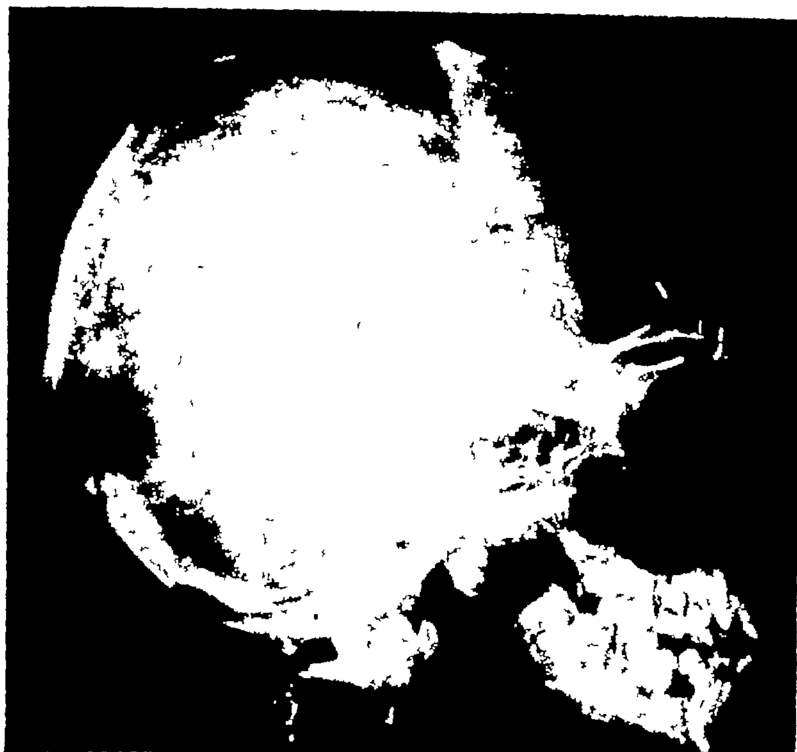


Fig 30 Case 5 Old skull defect resulting from compound skull fracture

new bone formation A plastic repair was therefore advised The day previous, a large piece of bone was removed for decompressive purposes from a patient with an inoperable glioma of the brain This was boiled with the instruments and used to repair the defect in the manner already described The patient was discharged on the twelfth postoperative day in good condition (Fig 31) An x-ray film taken six months later showed slight absorption of the lower border of the bone graft (Fig 32) but the defect was solid and did not pulsate The patient remained symptom-free and he was no longer forced to restrict his activities because of the skull defect



FIG. 31 Case 5 Appearance of wound twelve days after repair of old skull defect



FIG. 32. Case 5 X ray taken six months after repair of old skull defect Note slight absorption of the lower border of the graft.

Chapter VIII

EXTRADURAL HEMORRHAGE

Intracranial hemorrhages occurring between the inner table of the skull and the dura mater are known as extradural or epidural hemorrhages (Fig 33) and are a frequent cause of death following head trauma As early as 1750, J Hall operated for bilateral extradural hemorrhage, and reported recovery of the patient In 1751 he operated on a second case successfully Jacobson, in 1885, collected 70 cases of this type of injury and analyzed the symptomatology. In recent years, many cases have been reported from both surgical and neurosurgical clinics

Extradural hemorrhages are usually caused by tearing of the middle meningeal vessels or one of their main branches The tear results in an extravasation of blood between the dura mater and the skull, and as the collection of blood increases, the dura mater becomes separated from the bone and is compressed against the brain With the peeling of the dura mater from the skull, fresh vascular channels are opened and additional bleeding ensues In some cases the artery has been found torn just at the foramen spinosum, and in others the laceration has been in the main trunk of the meningeal artery Most often, however, the tear is either in the anterior or posterior division, or in both. A few cases of bilateral extradural hemorrhages due to laceration of branches of the middle meningeal arteries on both sides have occurred. Often the site of bleeding cannot be discovered either at operation or autopsy (Fig 34) Extradural bleeding occasionally occurs

from a rent in the dural sinuses or veins, the blood entering the extradural space through a tear in the dura mater itself The size of the clot in extradural hemorrhage varies from

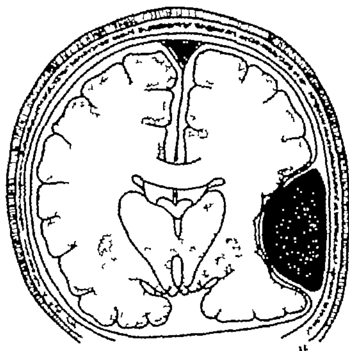


Fig. 33. Diagram to show position of extradural hemorrhage.

a very small one, weighing a few grams and having little importance in the symptoms or signs, to larger clots weighing as much as 250 or 300 Gm The usual clot weighs from 90 to 160 Gm At first the clot is semifluid and of the consistency of currant jelly It is nearly always very dark in color and may be almost black In the earliest stage it is easily freed from the dura mater but in cases which come to operation or autopsy after a day or longer it is very firm and adherent to the dura mater In a series of 504 autopsies in which the cranial bones were broken by external violence LeCount and Apfelbach found extradural hemorrhages in 199 In 104 cases the hemorrhage was large enough to have caused

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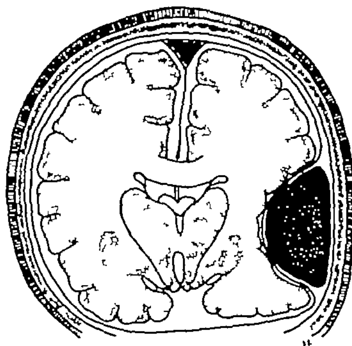


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serious compression of the brain Most of the large epidural clots are found with middle fossa fractures, although some are found in fractures of the posterior fossa, as well as in



Fig 34 Extradural clot disclosed at autopsy

fractures of the vault Occasionally, frontal fossa fractures are accompanied by extradural hemorrhage Not infrequently, no evidence of fracture can be found at operation or necropsy in patients with extradural hemorrhage due to tearing of the middle meningeal artery or its branches The clot most often covers the dura mater over the temporal, parietal, and occipital lobes, bilateral clots are encountered rarely In about one-fourth of the cases the clot compresses the dura mater over the occipital lobe, in rarer instances the frontal lobe compressed In about 40 to 50 per cent of the cases the brain is damaged merely by compression, and the cortex shows little or no evidence of contusion In some cases the brain has been contused sufficiently to cause a small amount of subdural bleeding In about one-fourth of the cases, extensive contusion and laceration of the brain is found associated with a large extradural clot, in these, the extra-

dural hemorrhage is usually undiagnosed Extradural hemor rhage is frequently found with relatively slight injury to the skull Indeed in some cases the injury has been trivial the head may have been bumped against a wall or injured in a minor fall, as in the following case

Case 6 A girl, 18 years of age fell down four steps to a land ing She did not seem seriously injured, but came to the emer gency ward because of slight bleeding from the left ear While being examined her speech became somewhat confused and she was admitted to the hospital Within thirty minutes she became deeply comatose and the pulse rate dropped to 40 At this time the blood pressure was 130/80 the left pupil was larger than the right, but both reacted to light Fifteen minutes later both pupils were dilated and fixed, the pulse rate was still 40 the blood pressure 138/80 On painful stimulation the patient pulled the left corner of the mouth up while the right side remained motion less The reflexes on the right side were depressed, and there was a marked Babinski sign on the right. A diagnosis of left extradural hemorrhage was made and the patient was operated upon with out delay A large extradural clot the result of a tear in the main trunk of the middle meningeal artery was found and removed The patient made an uneventful and complete recovery When examined two years later she was perfectly well An x ray film taken at the time of discharge shows the position of the bone defect. A silver clip used to control hemorrhage from the middle meningeal artery indicates the site where the vessel was torn (Fig 35)

In our experience, injuries incurred at work or play have been responsible for most of the cases Several were football and baseball accidents, the player being struck in the tem poral region by a ball or bat Direct injuries have been more often responsible for middle meningeal hemorrhage than injuries which occurred when the skull was in rapid motion at the time of the impact (automobile accidents) Of the re- ported cases about three-fourths were males Extradural

hemorrhages are uncommon in infancy and early childhood, as evidenced by the rarity of their reports in the medical literature The elasticity of the cranial bones, the relative

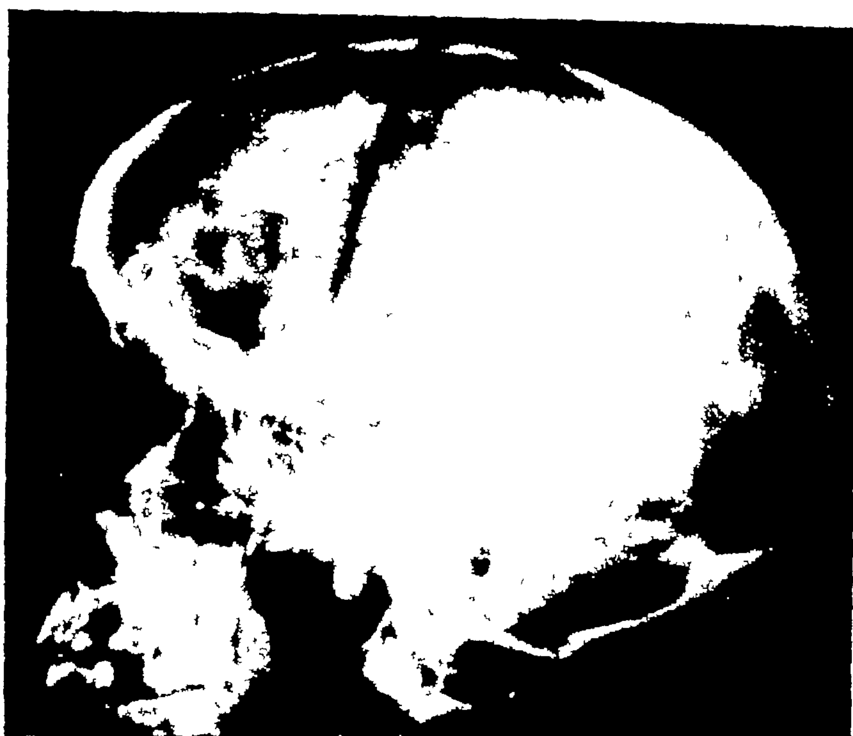


Fig 35 Defect in skull following operation for extradural hemorrhage Note silver clip in relation to groove for the posterior branch of the middle meningeal artery

absence of diploic spaces, the absence of well-marked grooving for the meningeal vessels, and the firm adhesion of the dura mater to the base of the skull in infancy and early childhood are the reasons why rupture of the meningeal artery occurs infrequently in this age period

In old age, too, the dura mater is firmly adherent to the entire skull and for this reason the incidence of extradural hemorrhage is quite rare in the aged.

Most cases occur during the active periods of life when exposure to direct head trauma is most common In every series

of cases studied the vast majority of patients have been between the ages of 20 and 50

In reports of craniocerebral injuries from clinics throughout the country the incidence of epidural hemorrhage is about 2.5 per cent However the frequency with which the medical examiner finds an epidural clot in persons found dead hours after relatively trivial head trauma indicates that the incidence of this condition is probably much higher

In the oft repeated clinical description of the symptomatology three phases have been described

1 Brief clouding of consciousness following the trauma

2. A period of relative well being which may last from one-half to several hours and in exceptional cases from two to three days This is commonly referred to as the "lucid interval" or "free period"

3 The period of cerebral compression with its headache vomiting drowsiness increasing to stupor and deep coma, concomitant with contralateral hemiplegia and convulsive phenomena During this period the loss of consciousness is accompanied by stertorous breathing Terminally, the picture is one of failing compensation with vasomotor collapse There is deep coma, the breathing is Cheyne-Stokes in character the patient is cold and clammy and decerebrate rigidity with tonic convulsions may precede the end.

Clinically there are great variations in this classical picture Initial clouding of consciousness may be entirely lacking On the other hand, when the trauma is severe enough to cause much cerebral damage, the primary period of unconsciousness may be prolonged throughout the period of the development of the clot and coma may be superimposed on the initial unconsciousness without any lucid interval A well marked lucid interval of varying duration is present in about 50 per cent of the cases In another 15 per cent the lucid in

terval is present but not marked, and in the remaining 35 per cent there is no lucid interval, due to concomitant brain damage or alcoholism.

In the typical clinical picture of epidural hemorrhage the signs and symptoms closely correspond to the stages of brain compression produced experimentally In the first stage, that of circulatory compensation, there is mild compression of the brain, evidenced by headache In the second stage, venous stasis increases as the clot grows larger Headache becomes severe and there is slowing of the pulse, with or without elevation of the blood pressure If the extravasation of blood is rapid, this stage passes quickly into the third stage, that of advanced compression Bradycardia is marked, the pulse may be as low as 40 per minute and the blood pressure rises This is the stage in which neurological signs and symptoms develop With the increasing compression the patient becomes drowsy, the pupil on the side of the hemorrhage dilates, probably due to pressure of the clot on the oculomotor nerve Contralateral hemiparesis develops, usually involving first the face, and then the arm and leg There may be accompanying Jacksonian convulsive seizures on the paretic side

When the hemorrhage is on the left side, right-handed individuals usually become aphasic before stupor sets in. Pyramidal signs, that is, increased reflexes and abnormal plantar responses are found on the side contralateral to the clot.

The diagnosis of extradural hemorrhage is easily made when the classical signs and symptoms are present The history of a definite lucid interval, followed by progressive stupor during the first or second day following a head trauma makes consideration of an extradural hemorrhage imperative If, in addition, there is a unilateral dilatation of the pupil

and a contralateral hemiparesis, the diagnosis is obvious When a history of a lucid interval has not been obtained, a diagnosis of extradural hemorrhage cannot be made with any degree of certainty Often, though a brief lucid interval occurred, it was not observed, and its occurrence could not be verified in a comatose patient Also when a large and rapid extravasation occurs in the extradural space, the effects of severe cerebral compression may become manifest before recovery from the initial commotio cerebri has taken place If there has been concomitant contusion and hemorrhage of the brain with loss of consciousness the signs of compression due to an extradural hemorrhage may be masked. The following points are helpful Extradural hemorrhages occur more often from accidents suffered during play or work, or from direct blows in the temporal region, than from accidents incurred while the head is in rapid motion In our own experience and from our acquaintance with the literature, extradural hemorrhages are relatively infrequent following automobile accidents

When x ray examination of the skull shows a fracture line crossing the groove for the middle meningeal vessels or one of their branches or a comminuted fracture in the squamous portion of the temporal bone, the likelihood of damage to the meningeal vessels is increased

In a patient with signs of cerebral compression where the spinal fluid is clear and colorless and under increased pressure extradural hemorrhage must be considered On the other hand, bloody spinal fluid does not rule out an epidural hemorrhage since slight bruising of the brain is sufficient to cause subarachnoid bleeding We found bloody spinal fluid in about half of the patients who were subjected to lumbar punctures

While a unilateral dilatation of the pupil is an important

finding, and occurs in many cases of extradural hemorrhage. Its absence does not necessarily rule out hemorrhage On the whole, unilateral dilatation of the pupil occurs in approximately 75 per cent of the cases Patients with epidural hemorrhages, who are seen after severe cerebral compression of several hours duration, may have bilaterally dilated pupils, and in these patients the prognosis is poor even with operation, because the ability of the medullary centers to recover is inversely proportional to the duration of the compression

In our cases and in those reviewed in the literature the pulse rate has been slow On the other hand, one should not ignore the possibility of epidural hemorrhage because of a fast pulse when the other signs and symptoms point to such a diagnosis

The progressive rise in blood pressure described in cases of cerebral compression has not been observed clinically as often as has the slow pulse Frequently, the blood pressure does not fluctuate very much during the entire course of the development of symptoms

The state of consciousness in extradural hemorrhage is not uniformly that of deep coma It is true that the classical picture is of a lucid interval followed by progressively increasing coma, nevertheless, one occasionally finds that with increasing compression, the patient becomes drowsy, although easily aroused This state is often mistaken for natural sleep, especially if the period of observation coincides with the period when natural sleep is expected In this category belong the numerous patients who are found dead the morning after a supposedly trifling injury to the head, and in whom blood clots are discovered at autopsy The following case is an instance

Case 7 A girl, 9 years of age, fell and struck the back of her head while roller skating She was dazed, but after a minute or

two was able to go home alone At home she complained of head ache and began to vomit A physician was called and rest in bed was advised The child became stuporous in the evening but

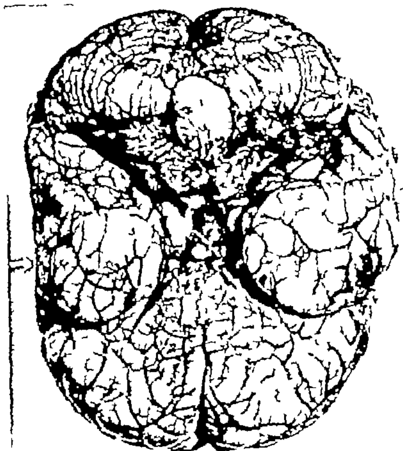


FIG. 36 Compression of the brain by an extradural hemorrhage. Note absence of cerebral contusion or laceration.

this was mistaken for sleep by the mother who did not become alarmed about the patient's condition until midnight, when she was finally brought to the hospital, where death occurred shortly after admission Autopsy disclosed a small linear fracture crossing the groove for the left middle meningeal artery and a large extradural clot on the left side. The brain was damaged only by compression (Fig 36) There was no subdural or subarachnoid bleeding and no contusion of the brain.

An alcoholic individual who remains stuporous beyond the time when one would expect him to recover from the effects of his recent drinking, must be kept under close watch because of the possibility of an injury sustained while he was intoxicated, with subsequent formation of a clot.

DIFFERENTIAL DIAGNOSIS

When an extradural hemorrhage is diagnosed and not found at operation, one may find a large collection of cerebrospinal fluid in the subdural space, the result of a tear in the arachnoid. These cases simulate extradural hemorrhage more closely than any other condition, but since an exploration is indicated in either case, treatment is similar to that for epidural hemorrhage. However, in cases of cerebrospinal fluid extravasations, the dura mater must be left open in order to allow for the absorption of the escaped cerebrospinal fluid, which does not take place from the subdural space.

Practically all severe lacerations of the brain are associated with more or less subarachnoid and subdural bleeding. These cases are due to severe head trauma and the signs of cerebral damage are profound from the onset. Acute subdural hemorrhage, when there has been minimal brain trauma and the hemorrhage results from a tear in a large cortical vessel, may simulate extradural hemorrhage. In these patients the signs of cerebral damage are progressive and the subdural bleeding compresses the brain in a manner similar to an extradural collection of blood. The treatment here, too, is operative, although the clot is not disclosed until the dura mater has been opened. As a rule, the development of symptoms is slower because the bleeding is venous rather than the rapid gush of blood which accompanies rupture of the middle meningeal artery.

Focal edema of the brain with the development of localizing neurological signs and symptoms which simulate a middle meningeal hemorrhage has been postulated by numerous observers This has been used to justify exploration when middle meningeal hemorrhage has been sought and not found and yet recovery has ensued It is possible that an extradural clot may be confined over the occipital or occipito-parietal area more rarely its situation may be far anterior The improvement following negative exploration can probably be attributed to the decompressive effects concomitant with opening of the dura mater This serves to prevent fatal cerebral compression With the tiding over of the critical period, life is sustained, and as the epidural clot slowly reabsorbs recovery takes place.

Rarely, cerebral thrombosis may be confused with middle meningeal hemorrhage especially if trauma preceded the onset of symptoms As a rule, however a definite lucid interval is not obtained the stupor coming on slowly and increasing gradually without an initial loss of consciousness The age of the patient the history of pre-existing arteriosclerosis or diabetes and the relatively slow progress of symptoms will, in most cases, serve to differentiate these two conditions X ray examination of the skull is of course, of great importance Nevertheless in an occasional case it may be impossible to differentiate thrombosis from middle meningeal hemorrhage except by exploration, as in the following patient

Case 8. A tailor aged 54 years fell and struck the right temporal region He was able to get up and walk home The next day he vomited and complained of dizziness The following day he became very drowsy and finally stuporous. He came under our care three days after the accident At this time he was semistuporous The pulse rate varied between 68 and 76. The blood pressure was 170/118. The patient had a left hemiparesis which became more marked during the period of observation Likewise,

An alcoholic individual who remains stuporous beyond the time when one would expect him to recover from the effects of his recent drinking, must be kept under close watch because of the possibility of an injury sustained while he was intoxicated, with subsequent formation of a clot

DIFFERENTIAL DIAGNOSIS

When an extradural hemorrhage is diagnosed and not found at operation, one may find a large collection of cerebrospinal fluid in the subdural space, the result of a tear in the arachnoid These cases simulate extradural hemorrhage more closely than any other condition, but since an exploration is indicated in either case, treatment is similar to that for epidural hemorrhage However, in cases of cerebrospinal fluid extravasations, the dura mater must be left open in order to allow for the absorption of the escaped cerebrospinal fluid, which does not take place from the subdural space

Practically all severe lacerations of the brain are associated with more or less subarachnoid and subdural bleeding These cases are due to severe head trauma and the signs of cerebral damage are profound from the onset Acute subdural hemorrhage, when there has been minimal brain trauma and the hemorrhage results from a tear in a large cortical vessel, may simulate extradural hemorrhage In these patients the signs of cerebral damage are progressive and the subdural bleeding compresses the brain in a manner similar to an extradural collection of blood The treatment here, too, is operative, although the clot is not disclosed until the dura mater has been opened As a rule, the development of symptoms is slower because the bleeding is venous rather than the rapid gush of blood which accompanies rupture of the middle meningeal artery

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a faint Babinski sign became definite during this period The pupils were equal and reacted promptly to light Lumbar puncture yielded a clear colorless fluid under a pressure of 120 mm of water X-ray examination of the skull did not reveal a fracture Because of the history of an injury in the right temporal region and the slowly progressive left hemiplegia, an exploration under local anesthesia was done, to rule out an extradural hemorrhage. An extradural clot was not found The patient suffered no ill effects as a result of the operation. The hemiplegia slowly improved and the sensorium cleared He left the hospital three weeks later walking with the aid of a cane The final diagnosis was "cerebral thrombosis."

TREATMENT

The treatment of extradural hemorrhage is operative Of all surgical emergencies, extradural hemorrhage is among the most urgent To delay operation after a diagnosis is made is to jeopardize the patient's chances of recovery, since with each hour of severe cerebral compression after the first two or three hours, the ability of the vital centers to recover diminishes rapidly. In 1885, Wiesman collected from the literature 257 cases of extradural hemorrhage One hundred and forty-seven were treated expectantly and all but 16 died —a mortality of almost 90 per cent The remaining 110 were operated upon and 90 recovered—a mortality of less than 33 per cent With modern methods and prompt surgical intervention the mortality in uncomplicated extradural hemorrhage can be reduced much further.

In most cases of extradural hemorrhage the usual subtemporal decompression incision is employed (Fig 37). The incision should extend to the zygoma so that if the artery has been torn at the foramen spinosum it can be exposed Usually when the temporal muscle has been separa[illegible] bone, a fracture will be found crossing the ch[illegible] geal vessels. As a rule, as soon as the [illegible]

broken through with the trephine or perforator the clot will ooze out of the opening The opening in the skull is then enlarged sufficiently to permit removal of the clot and ex

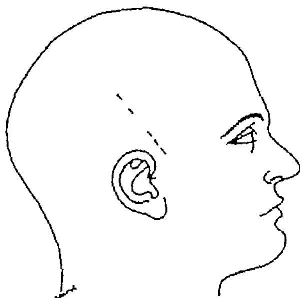


FIG. 37 Incision for subtemporal decompression This exposure is adequate for most extradural hemorrhages.

posure of the torn vessel If the artery has been severed at the foramen spinosum it is necessary to remove the bone as far down as the floor of the middle cranial fossa The fora men spinosum can then be plugged with a small pellet of cotton impregnated with bone wax or a properly prepared orangewood peg If the vessel has been torn on the dura mater it can be secured with a silver clip or a fine silk liga ture If a ligature is used care must be taken not to injure the underlying brain and cortical vessels When all the clot has been removed the compressed brain in favorable cases, expands and begins to pulsate If the brain fails to expand normal saline solution given rapidly, intravenously may aid. Occasionally a retained clot posteriorly may prevent expan sion of the brain The wound should not be closed until the

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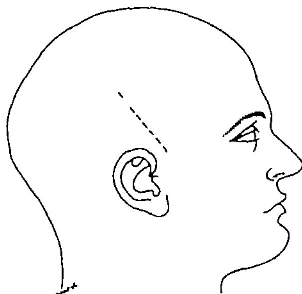


FIG. 87 Incision for subtemporal decompression This exposure is adequate for most extradural hemorrhages.

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brain has expanded properly. As a rule, it is not necessary to open the dura mater. If there is any evidence, however, that an associated subdural hemorrhage has occurred, the dura mater should be opened without hesitation. Operation for extradural hemorrhage is preferably done under local anesthesia, and in favorable cases, when the clot has been removed, the patient becomes more alert while on the operating table. It is wisest to place a small rubber tube drain on the dura mater, with its exit at the lower corner of the wound, which is then carefully closed in layers with interrupted silk sutures.

In the postoperative care all measures used in the management of any head injury are employed. Narcotics are prohibited since they depress the medullary centers. If sedation is required the barbituric acid derivatives, paraldehyde, chloral, and bromides are preferred. It is not necessary to restrict fluid, since as a rule there has been little damage to the brain except compression by the clot. Patients are kept in bed for three weeks, although in favorable cases most patients feel perfectly well a few days after the operation.

In uncomplicated cases of extradural hemorrhage operated upon within the first few hours after the injury, the outlook for complete and prompt recovery is excellent. When operation has been delayed the mortality increases rapidly, so that after deep coma has existed for more than a few hours the chances of recovery are very poor. If bilaterally dilated fixed pupils, or decerebrate rigidity exists, the patient rarely recovers. Nevertheless, an occasional case has been saved even when these signs were present. A preoperative elevation of temperature above 103° F. is a grave omen.

There are usually no sequelae in patients promptly operated upon. The defect in the skull is low down, adequately covered by temporal muscle and fascia, and this practically

never causes any symptoms In the patients whom we have observed there have been none of the post-concussion symptoms which frequently follow severe head injuries An occasional patient operated upon after compression has existed for several hours may be left with extensive neurological disturbances

Case 9 A boy 5 years of age was operated upon elsewhere after a severe compression of the brain by an extradural clot had existed for a few hours He had decerebrate rigidity at the time of operation but nevertheless recovered. Signs of damage to the midbrain persisted, and after three years when he came under our observation he still had spasticity of all four extremities and dysarthric speech.

Chapter IX

TRAUMATIC SUBDURAL HEMATOMA

A subdural hematoma is an encapsulated collection of fluid or clot in various stages of disintegration, located in the space between the dura mater and the arachnoid membrane (Fig 38) Until recently there was considerable confusion concerning subdural hematomas The relation of trauma to subdural hematoma was not clearly indicated until the publication of a paper in 1914 by Wilfred Trotter Following a report by Putnam in 1925, interest was stimulated, and since then there have been many excellent contributions, and a clearer understanding of subdural hematoma At first most of the cases were operated upon in neurosurgical clinics, as many of the patients were suspected of harboring brain tumors In the past few years, however, subdural hematoma following injury has become well known to neurologists, surgeons, and the medical profession in general, so that many cases are operated upon every year The historical aspects of the condition have been admirably covered in the report of Putnam

Traumatic subdural hematomas occur more frequently in males than in females, in our own series (Table II) males were affected about twice as often as females This lesion occurs in all age periods, from infancy to senescence, although the average age is about 40 With rare exceptions a history of trauma to the head can be obtained However, in many cases the injury is considered trivial, and is often forgotten by the patient until the traumatic origin of his illness is

stressed Fracture of the skull is not usually found in these patients

The rôle of toxic factors in the etiology of subdural hema

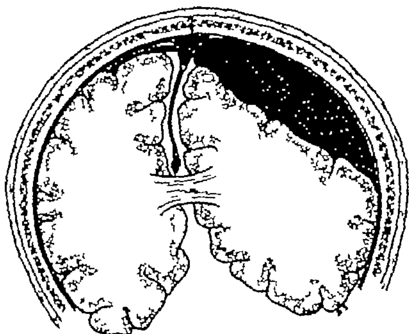


FIG. 39 Diagram of subdural hematoma. Note location of the clot between the inner surface of the dura and the arachnoid. The falx is displaced. A cortical vein is seen entering the longitudinal sinus. These veins are relatively unsupported and enter the sinus at right angles. They may be torn by even slight trauma.

toma is not clear, though a history of alcoholism is frequently obtained in these patients

Subdural hematomas result from bleeding into the subdural space (Fig 39) The source of the bleeding is usually a tear in one of the cerebral veins entering the dural sinuses The cerebral veins entering the lacunae of the superior longitudinal sinus are extremely thin walled unsupported short, and without tortuosity One end of this unsupported portion of the vein is fixed to the rigid dural sinus while the other is attached to the moveable cerebral hemisphere An injury

to the head from the application of a force to the frontal or occipital region produces a movement of the brain without any marked alteration in the position of the dura mater This

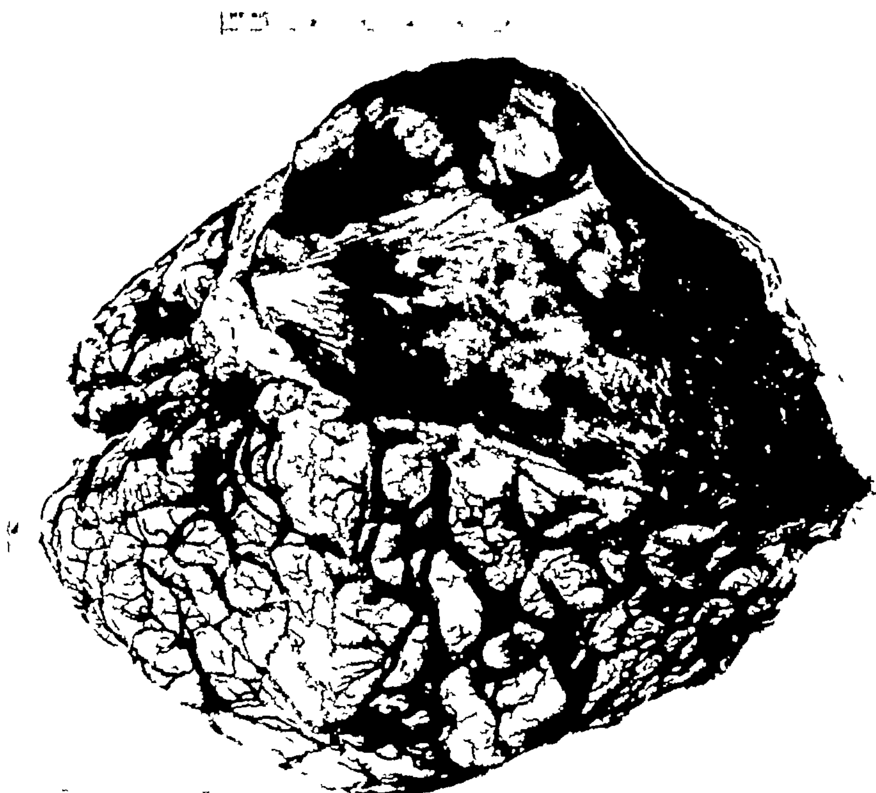


Fig 39 Traumatic subdural hematoma This patient suffered a mild head injury four weeks previously His family withheld consent for operation

may cause a tear in a cerebral vein, usually at its rigid attachment to the dural sinus The pial and subarachnoid portion of the vein is more tortuous and mobile, and is not as apt to rupture from this type of trauma The rigidly attached portion, which does tear, is extra-arachnoid and consequently the blood escapes from the vessel into the space between the arachnoid membrane and the dura mater The blood coagulates rapidly and soon becomes encysted The surface of the clot nearest the dura becomes covered by a membrane

of fibroblasts and connective tissue which may be from 1 rr to as much as 5 mm thick depending on its age while the surface nearest the arachnoid membrane is of transparent thinness and made up of a layer of arachnoidal cells When bleeding ceases, and the clot is entirely enclosed by membrane, there are no facilities for its absorption If the amount of bleeding is considerable at the outset, symptoms develop relatively rapidly, whereas, when the encysted clot is small at the beginning symptoms may be delayed for several weeks or months As the clotted blood within the membranes disintegrates into simpler chemical compounds the osmotic tension is altered so as to favor the passage of fluid into the cyst, and as the mass enlarges symptoms develop and progress In addition the hematoma increases in size due to recurrent bleeding from many newly formed capillaries in the outer membrane In the early stages the contents of the hematoma is semifluid dark brownish red in color and jelly like in consistency Later as the hemoglobin molecules break down the cyst contains a yellowish green fluid of high protein content Histologically the outer membrane consists of fibrous tissue with many young fibroblasts and newly formed capillaries At times there may be an infiltration with polymorphonuclear cells, lymphocytes and plasmacytes The inner membrane is composed of a proliferation of the arachnoidal cells A few cases in which calcification has occurred in hematomas of long standing have been reported

The symptomatology of subdural hematoma varies considerably depending on the severity of the initial trauma, and the size of the initial extravasation into the subdural space Most cases result from relatively minor injuries and often the details of the initial injury are forgotten or unknown Occasionally an initial brief period of unconsciousness is present followed often by a period of relative well being

"the latent interval," lasting from a few days to several weeks or months, but averaging about twenty-five days. During this period the patient seems to be in good health, with the exception perhaps of an occasional headache. In some patients headache occurs soon after the injury, but is unaccompanied by other symptoms until disturbances in the state of consciousness occur. The patient then becomes drowsy. When roused he complains of severe headaches. Poor memory, confusion, irritability, and other mental symptoms may also be present. Some of the patients in our series had generalized or Jacksonian motor convulsions, and others developed a hemiparesis.

Failing vision is the presenting symptom in some patients with subdural hematoma, who are suspected of having brain tumors. The variability, and irregular development of the symptoms are characteristic of subdural hematoma.

The state of consciousness in patients with subdural hematoma varies from complete awareness to deep coma. When the symptoms develop rapidly, after a period of somnolence of two or three weeks, the patient may suddenly become deeply comatose. On the other hand, some patients are never comatose, but come under observation because of failing vision and headaches. Most of our patients were very drowsy or stuporous. They could be roused, but were irritable, and had to be coaxed to answer questions.

Rand noted a high-pitched note on percussion of the skull in every case in a series of seven. The pulse rate is slow in many of the cases. In our own series the blood pressure shows no characteristic alteration. Meningeal irritation as evidenced by stiff neck and a positive Kernig sign is occasionally present. Kaplan noted especially the presence of a unilaterally dilated fixed pupil on the side of the hematoma. In other series of cases the state of the pupils has not been particu-

larly characteristic. About 75 per cent of the patients had changes in the eye grounds, although marked papilledema was present in only about 25 per cent of our cases and in 40 per cent of the cases collected by Jelsma In most of the patients, the visual fields could not be examined because of the mental state of the patient When fields were obtained they were usually normal, although homonymous hemianopsia has been reported

Reflex changes, pyramidal tract signs, and paralyses are frequently found in patients with subdural hematoma Such findings are notoriously unreliable in localizing the side of the lesion since the massive displacement of all the midline structures of the brain may cause pressure on the opposite cerebral peduncle against the unyielding incisura tentorii, so that pyramidal signs and paralyses may be found on the side of the lesion Bilateral pyramidal tract signs have been found in a number of patients with unilateral subdural hematoma Changes in sensation were not noted frequently but most patients with subdural hematomas are poor subjects for sensory examinations because of their mental state Aphasia is infrequent X ray examination of the skull is not of great aid in diagnosis Fractures are infrequent in subdural hematoma and signs of increased intracranial pressure, such as erosion of the sella turcica and convolutional impressions are absent in most cases

In about 20 per cent of the cases the cerebrospinal fluid is xanthochromic and under increased pressure but in most the fluid is clear and the pressure within normal limits or only slightly elevated The cell count is usually normal though occasionally a pleocytosis is found The total protein content of the fluid is usually slightly elevated 50 to 75 mg per cent when determined by the Dennis Ayer method Table II

T

FINDINGS IN 15 CASES OF TRAU

Case	*Sex*	*Age*	*Injury*	*Onset of Symptoms*	*Time of Operation*	*Headache*	*Stupor*	*Pu*
1	F	48	Blow to head	Unknown	5 wks	Not known	Marked	Left dila
2	M	25	Struck on jaw	12 days later	7 wks	Severe	None	Nor
3	M	45	Kicked in head	Immediate	2 wks	Severe	Deep coma	Rig dila
4	M	44	Blow to head	2 wks	3 wks	Severe	Marked	Nor
5	F	49	Fell on head	3 days	8 days	Not known	Marked	Nor
6	M	52	Struck by automobile	Immediate	8 days	None noted	Marked	Nor
7	M	38	Fist fight	Not known	2 wks	Not known	Marked	Rig dila
8	M	52	Fell to sidewalk	1 wk	4 wks	Marked	Marked	Nor
9	M	14	Fell in bathtub	Immediate	3 mos	Modererate	None	Nor
10	M	44	Not known	Not known	2 mos	Marked	Deep coma	Sma equ
11	F	50	Fell down stairs	9 days	9 days	Moderate	Marked	Left larg
12	F	42	Struck by automobile	4 days	17 days	Moderate	Marked	Rigl dila
13	M	48	Struck by automobile	2 wks	2 mos	Severe	Marked	Equ
14	F	73	Fell, struck head	3 days	9 days	Severe	Marked	Equ
15	M	60	Fell down stairs, struck head	2 wks	5 wks	Slight	Drowsy	Left larg

II

Subdural Hematoma Operated Upon

Fundi	*Focal Signs*	*Pulse Rate*	*Spinal Fluid*	*Spinal Fluid Pressure*	*X-ray of Skull*	*Side of Lesion*	*Result*
Blurred discs	Right hemiparesis	60	Clear and colorless	Slightly increased	Negative	Right	Cured
Choked discs	None	80	Clear and colorless	500 mm. of water	L. ventricle displaced	Left	Cured
Normal	Right side spastic	50	Clear and colorless	180 mm of water	Negative	Left	Died
Blurred discs	Left hemiplegia	40	Clear and colorless	140 mm. of water	Negative	Right	Cured
Normal	Reflexes increased on left	64	Xanthochromic	70 mm. of water	R. temporal fracture	Left	Died
Normal	Left hemiplegia	72	Bloody	Slightly increased	Negative	Right	Cured
Normal	Left hemiparesis	56	Clear and colorless	260 mm. of water	Negative	Right	Died
Early choking	Reflexes on left increased	66	Xanthochromic	Slightly increased	Negative	Right	Died
Choked discs	None	72	Clear and colorless	380 mm. of water	Ventricular shift to R.	Left	Cured
Normal	None	50	Clear and colorless	110 mm. of water	Negative	Bilateral	Cured
Normal	Left hemiparesis	100	Xanthochromic	Increased	R. temporal fracture	Left	Cured
Normal	Left hemiparesis	56	Xanthochromic	80 mm. of water	Negative	Right	Cured
Normal	Left hemiplegia	58	Clear and colorless	340 mm of water	Negative	Bilateral	Cured
Normal	Right hemiplegia	96	Clear and colorless	120 mm. of water	Negative	Left	Cured
Normal	Right hemiparesis	72	Clear and colorless	150 mm. of water	Pineal gland shifted to right	Bilateral	Cured

TABLE

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11	F	50	Fell down stairs	9 days	9 days	Moderate	Marked	Left large
12	F	42	Struck by automobile	4 days	17 days	Moderate	Marked	Right dilated
13	M	48	Struck by automobile	2 wks	2 mos	Severe	Marked	Equal
14	F	73	Fell, struck head	3 days	9 days	Severe	Marked	Equal
15	M	60	Fell down stairs, struck head	2 wks	5 wks	Slight	Drowsy	Left large

II

SUBDURAL HEMATOMA OPERATED UPON

Fundi	*Focal Signs*	*Pulse Rate*	*Spinal Fluid*	*Spinal Fluid Pressure*	*X-ray of Skull*	*Side of Lesion*	*Result*
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Normal	Right hemiparesis	72	Clear and colorless	150 mm. of water	Pineal gland shifted to right	Bilateral	Cured

gives the important data in the history, symptomatology and findings in our own cases

The diagnosis of subdural hematoma presents few difficulties, when a history of trauma is followed in a few weeks or months by headache, mental changes, drowsiness, slow pulse and a unilaterally dilated and fixed pupil This combination of symptoms and signs is found in only a small proportion of the cases However, some of them have been present in nearly every case Often it is not possible to exclude a brain tumor except by exploration The history of trauma and the absence of changes in skull roentgenograms are in favor of subdural hematoma The spinal fluid pressure in brain tumors is usually definitely elevated, while in subdural hematoma it is normal or only slightly elevated

Post-traumatic encephalopathy rarely presents the drowsiness, mental changes, or slow pulse In addition, the initial trauma is usually much more severe, and the symptoms remain more or less constant, whereas in subdural hematoma the symptoms generally become progressively more severe, although for brief periods there may be marked fluctuations in the patient's well-being

When a subdural hematoma is suspected it should be searched for and evacuated as soon as possible Patients operated upon before deep coma supervenes have an excellent chance for complete restoration of health On the other hand, those operated upon when in deep coma rarely recover The type of operative procedure depends on the age of the hematoma and the condition of the patient. Hematomas which are mostly fluid can be removed through a trephine opening, while those which are coherent are best dealt with through a larger opening in the skull

If the patient is in relatively poor condition, and semicomatose or comatose at the time of operation, we believe in

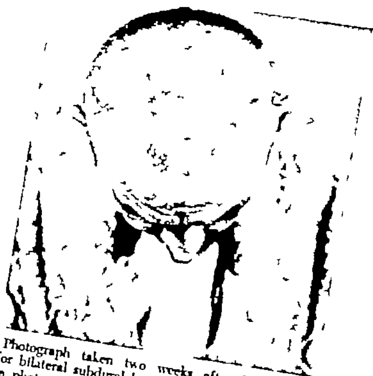


FIG. 40 Photograph taken two weeks after bilateral transtemporal trepanation for bilateral subdural hematomas. The scars are practically invisible in the photograph

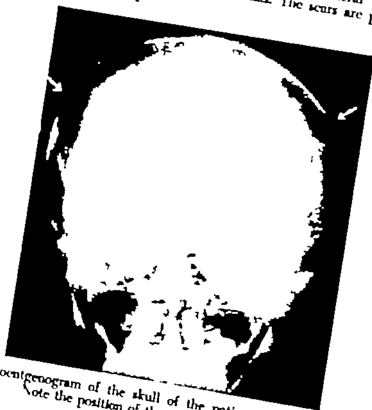


FIG. 41 Roentgenogram of the skull of the patient shown in Figure 40 Note the position of the openings in the skull.

making bilateral openings in the skull in the upper temporal regions (Figs 40, 41). As a rule, as soon as the dura mater is exposed, the presence of a subdural hematoma is disclosed by the plum-blue color shining through When the dura mater is incised the thick, velvety outer membrane of the hematoma is reached, and after this is opened the contents can be removed Fluid hematomas are easily dealt with. With the escape of the contents of the hematoma, the brain expands and fills up the cavity between the dura mater and the surface of the brain The expansion of the brain can sometimes be facilitated by the intravenous administration of normal saline solution. As much of the thick outer membrane as can be conveniently removed, without enlarging the opening in the skull too much, should be teased out, although there is no evidence that the retained membranes are harmful Often a semicomatose patient regains consciousness while on the operating table Since bilateral clots are present in about 10 to 25 per cent of the cases it is always safest to explore both sides

When a solid or coherent hematoma is found, a larger opening in the skull is required The usual subtemporal decompression may be used, although a small bone flap is preferable, if the condition of the patient permits, since it avoids the disadvantages of a large defect in the skull. When the diagnosis of subdural hematoma remains doubtful, it is advantageous, providing the patient is in fairly good condition, to make bilateral openings in the posterior parietal region, so that if a hematoma is not found, a ventriculogram can be made and the lesion localized

Fleming and Jones, Frazier, and others advised two openings on each side, one in the frontal and one in the parietal region so that the hematoma could be washed out This has not been necessary in any of our cases, since with fluid hema-

tomas the contents are easily evacuated through a single opening in the skull, while organized hematomas require either a bone flap or a subtemporal decompression for their removal

The mortality in patients who are deeply comatose at the time of operation is high while those with subdural hematomas who are operated upon before deep coma supervenes usually make excellent recoveries

Following operation headaches are relieved at once Two of our patients with intolerable headaches operated upon under local anesthesia said that their headaches disappeared immediately after the outer membrane was incised, allowing the fluid contents of the hematoma to escape There have been few untoward sequelae Some patients have had mild headaches and dizziness but most of them have been restored to normal health The following case histories illustrate many of the points indicated in the preceding pages

Case 10 A colored female aged 48 years, was admitted to the hospital because of stupor A history was not available Both eyes were deviated to the right, there was a right hemiparesis with a right Babinski sign. The left pupil was larger than the right both reacted poorly to light. The nasal margins of both optic discs were blurred. The pulse rate was 60 the blood pressure 160/100. X-ray examination of the skull was negative. Twelve hours after admission the stupor had increased to deep coma Spinal puncture yielded a clear colorless fluid under slightly increased pressure which showed 10 lymphocytes per c.mm and a slight increase in globulin Bilateral trephine exploration in the frontotemporal regions was done A large subdural hematoma containing yellowish syrupy fluid was found on the right side Following its evacuation the patient made an uneventful recovery After the operation the patient gave a history of having received a blow with the fist to the head five weeks previously

Case 11 A white male waiter 25 years of age was admitted

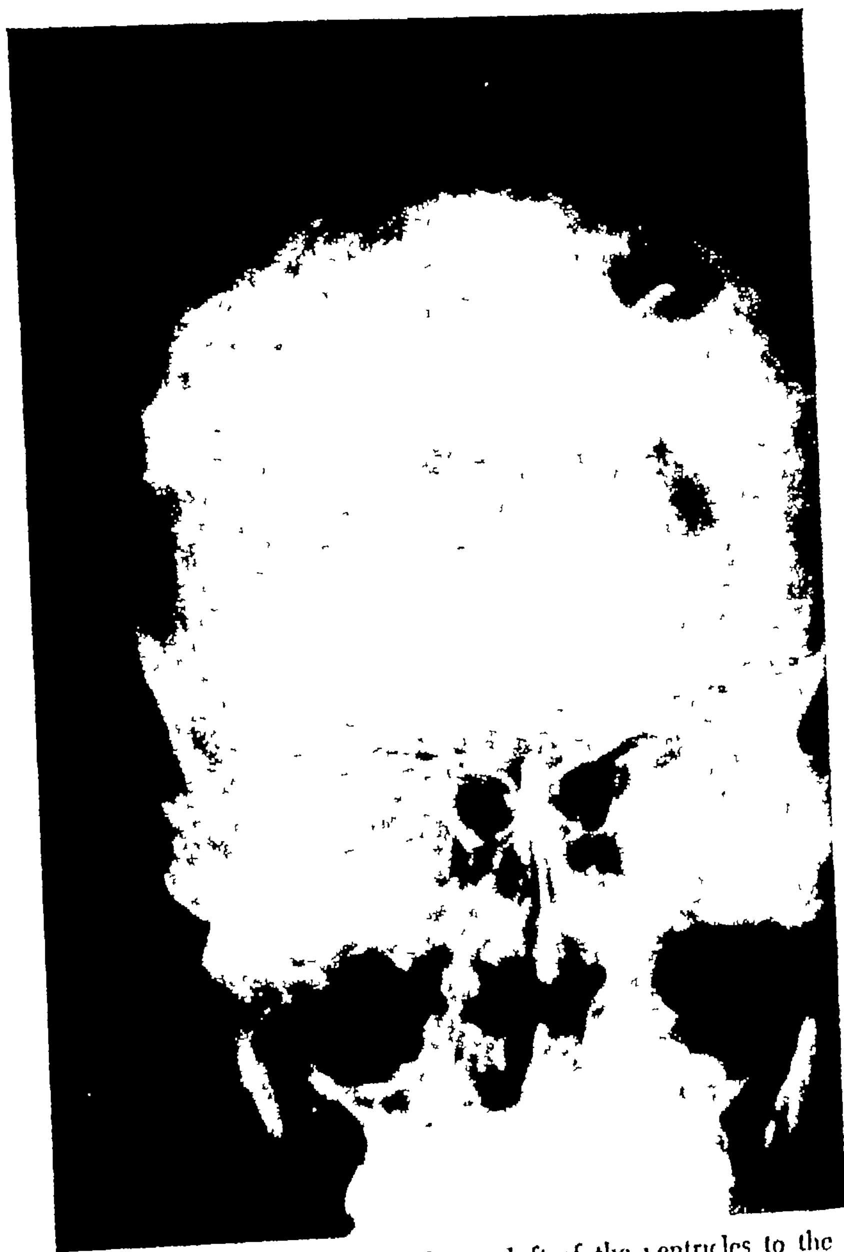


FIG 42 Case 11. Ventriculogram shows shift of the ventricles to the right and depression of the body of the left lateral ventricle

to the hospital because of occipital headaches vomiting and failing vision One month previously he had received a blow to the jaw and was "knocked out" for a few minutes However he con

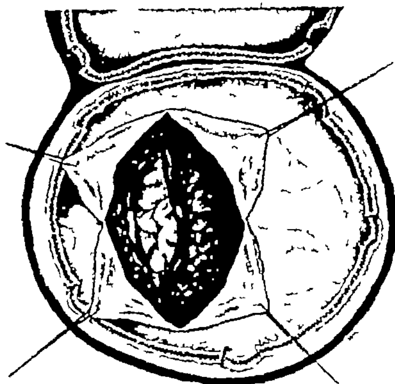


FIG. 43. Case 11 Traumatic subdural hematoma disclosed by bone flap Such an extensive procedure is rarely necessary

tinued at work for twelve days when severe occipital headaches forced him to stop

Examination revealed bilateral papilledema and a slight right central facial weakness Cerebrospinal fluid obtained by lumbar tap was clear and colorless under a pressure of 500 mm of water Plain x ray examination of the skull was negative However x ray examination after the injection of air by ventricular puncture showed a shift of the entire ventricular system to the right (Fig 42) In addition the body of the left lateral ventricle was depressed. A left parietal craniotomy disclosed a typical subdural hematoma (Fig 43) and its removal was followed by rapid and complete recovery

Case 12 A white male, 44 years of age, was admitted to the hospital in deep coma Two months previously he began having severe headaches, accompanied by vomiting After two weeks he was forced to quit his job as a chef He gradually became more and more lethargic, and his memory became markedly impaired. A few days before admission he became drowsy, and finally comatose Except for the deep coma, the neurological examination was essentially negative The fundi were normal Spinal tap yielded a clear colorless fluid under a pressure of 110 mm of water The pulse rate varied between 50 and 60. Frontal ventriculography was done, but the ventricles failed to fill properly, affording no assistance in diagnosis Lumbar encephalography was likewise unsatisfactory Bilateral posterior parietal trephine exploration disclosed a bilateral subdural hematoma Its evacuation was followed by rapid recovery, although during the first few days after operation the patient was markedly confused and disoriented His mental condition eventually cleared up entirely, and when examined at the time of discharge he was fully oriented and had normal insight

SUBDURAL HEMATOMA IN INFANCY AND EARLY CHILDHOOD

Subdural hematomas in infancy and early childhood differ somewhat in several important factors from those in adults A history of trauma is rarely obtainable, although birth injury is always possible except in infants delivered by elective Cesarian section

Subdural hematoma should be suspected in an infant whose head, normal in size at birth, begins to enlarge progressively several weeks to several months later (Fig. 44) Sherwood found enlargement of the head, vomiting, irritability, retinal hemorrhages and convulsions, together with malnutrition in many of his cases

Because of the enlargement of the head, many of these cases have been diagnosed as "hydrocephalus" This condition can be ruled out by bilateral subdural aspiration through

the lateral angles of the anterior fontanelle Care should be taken not to puncture too deeply since both walls of the encapsulated hematoma may be penetrated, resulting in an



FIG. 44 Bilateral subdural hematoma in an infant. This patient received a blow to the head in early life At the age of four months she had a convulsion. After this there was progressive enlargement of the head and a right hemiparesis. Fluid obtained by lumbar tap was clear but fluid from the subdural space obtained by fontanelle puncture was chocolate color The fundi showed old hemorrhages and exudates. The patient died at the age of eight months while being studied in the hospital.

erroneous diagnosis The fluid from a subdural hematoma in infancy is usually xanthochromic or bloody

In general, the treatment of subdural hematoma in infants has not been as satisfactory as in adults, since infants tolerate poorly the sudden alteration of intracranial physiology subsequent to the evacuation of a hematoma Naffziger and Brown have employed bilateral trephine openings, whereas Peet and Kahn prefer an osteoplastic flap Recently Ingraham and Heyl advocated the following plan for the treatment of subdural hematomas in infancy and early childhood

1 Lumbar puncture with the removal of not more than

10 c c of fluid If blood is present in the cerebrospinal fluid, a daily spinal tap should be done until the fluid is clear.

2. Bilateral subdural taps through the coronal suture. The scalp should be carefully shaved and aseptic technique used The puncture is made with a short needle having a short bevel, and well away from the midline to avoid injury to the superior longitudinal sinus If a hematoma is present there will be a free escape of a yellow or blood-tinged fluid Subdural taps are repeated daily on alternate sides for a week or ten days During this period the infant is built up by careful feeding, parenteral fluids and transfusions

3 Bilateral burr holes are made. They are placed so that they may later be included in a bone flap At this operation the fluid-containing hematomas can be emptied completely. If desired, air may be introduced at this time so that x-ray films can show the extent of the lesion If a solid clot is present it may be partially washed out If definite membranes are present the fourth step is indicated

4 A bone flap is turned down in the frontoparietal region, and as much as possible of the membranous envelope is removed If a membrane is known to be present over both hemispheres, a second bone flap is done whenever the patient's condition permits, usually one or two weeks later.

Applying the above plan, Ingraham and Heyl reported 10 recoveries and 1 death in a series of 11 patients, 10 of whom were from 2 to 13 months of age, and one 6 years.

ACUTE SUBDURAL HEMORRHAGE

With every severe craniocerebral trauma, acute subdural hemorrhage occurs to some extent This is nearly always associated with contusion and laceration of the brain, and hemorrhage from the cortical veins (Fig. 45). The subdural bleeding results from tears in the arachnoid membrane, which permit the subarachnoid hemorrhage to enter the subdural

space In these cases the clinical picture is usually predominantly one due to the severe cerebral injury, the subdural collection of blood playing a relatively small rôle in the cau

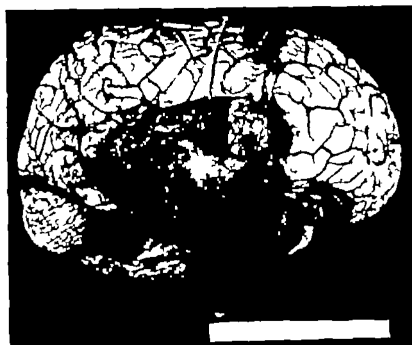


FIG. 45 Acute subdural hemorrhage associated with laceration of the brain

sation of the symptomatology However in some cases the subdural hemorrhage becomes extensive enough to give focal and general signs due entirely to its presence and these are manifest by insidious and progressive hemiplegia and deepening stupor Rarely as in the following case an acute subdural hemorrhage may simulate an extradural hemorrhage

Case 13 A boy 7 years of age fell from a slide while playing He did not lose consciousness at once When the ambulance surgeon arrived he found the patient walking around but unable to give his name or address. On the way to the hospital the patient became drowsy Examination disclosed a large hematoma in the left parietal region The pulse varied from 64 to 70 The blood pressure was 102/72. A lumbar puncture yielded a bloody fluid under a pressure of 400 mm of water



FIG 46 Case 13 Fracture in the left posterior parietal region associated with acute subdural hemorrhage



FIG 47 Case 13 Defect in the skull following operation for acute subdural hemorrhage Note the numerous silver clips which were used to ligate the bleeding cortical vessel

The patient graduall) became more and more drowsy and when seen seven hours after the accident he was in deep stupor A right hemiplegia was present, and a Babinski sign was found



Fig. 48. Case 13 Operative scar at time of discharge from the hospital.

on both sides The pupils were equal in size and reacted well to light. X ray examination of the skull revealed a fracture in the left posterior parietal region (Fig 46) A diagnosis of probable extradural hemorrhage was made and an exploration was carried out immediately A stellate fracture was found in the posterior parietal area. An opening about 3 cm in diameter was made in the skull A thin extradural hemorrhage was found. The dura was tense and through it a bluish discoloration was seen. Upon opening the dura an extensive subdural hemorrhage was disclosed. A torn cortical vessel, which was bleeding actively was found and ligated with silver clips (Fig 47) The patient gradually improved. The hemiplegia and aphasia persisted for several days but at the time of discharge from the hospital about one month after the injury the weakness had cleared up and there was practically no speech defect (Fig 48)

The patient with a cerebral laceration usually shows severe clinical signs from the beginning. As he is being observed, in the non-fatal case, the signs gradually disappear When the signs increase in severity continued bleeding must be suspected, and if progressive focal signs develop, bleeding may be subdural in location It is in these patients that surgical intervention is indicated These cases are practically always associated with fracture of the skull There has been some confusion between subdural hematoma and acute subdural hemorrhage because of laxity in terminology. From an etiological, pathological and clinical standpoint the two conditions differ greatly (Table III)

TABLE III

DIFFERENTIATION BETWEEN TRAUMATIC SUBDURAL HEMATOMA AND ACUTE SUBDURAL HEMORRHAGE

	Subdural Hematoma	*Acute Subdural Hemorrhage*
Trauma	Slight	Severe
Latent period	Several days to several weeks	None
Brain damage	Due to compression	Severe contusion and laceration
Source of bleeding	From cortical veins as they enter the dural sinuses Arachnoid intact	From lacerated cortical veins on the convexity with the blood entering the subdural space through tears in the arachnoid
Fracture of the skull	Rare	Very frequent

Chapter X

FRACTURE OF THE SKULL WITH INVOLVEMENT OF THE PARANASAL SINUSES, MASTOIDS AND MIDDLE EAR

Fractures of the skull with injuries to the nasal accessory sinuses and mastoids are logically grouped together From a clinical standpoint however there are many important differences They are similar in that they constitute a group in which the nature of the fracture determines the type of therapy in contradistinction to most head injuries where the trauma to bony structures is of secondary importance

The frontal sinuses because of their anatomical location are particularly prone to injury in head-on collisions or com bat When violence is applied to the forehead there is often fracture of the walls of the frontal sinuses (Fig 49) When the force is of greater severity the fracture frequently ex tends basally to the cribriform plate or to the roof of the orbit, and superiorly to the vault The dura mater over the frontal lobes is thin and that along the floor of the frontal fossa is thin and adherent to the bone Thus it is often torn when the closely applied bone is fractured and a portal is opened for the passage of infectious material from the nasal sinuses into the intracranial cavity Likewise an avenue for the escape of cerebrospinal fluid from the nose is established Fractures involving the frontal sinuses may be divided into four groups

1 Linear or depressed fractures without laceration of the scalp and without cerebrospinal rhinorrhea or pneumocephalus

2 Compound fractures without rhinorrhea or pneumocephalus

3 Frontal fractures with cerebrospinal fluid rhinorrhea

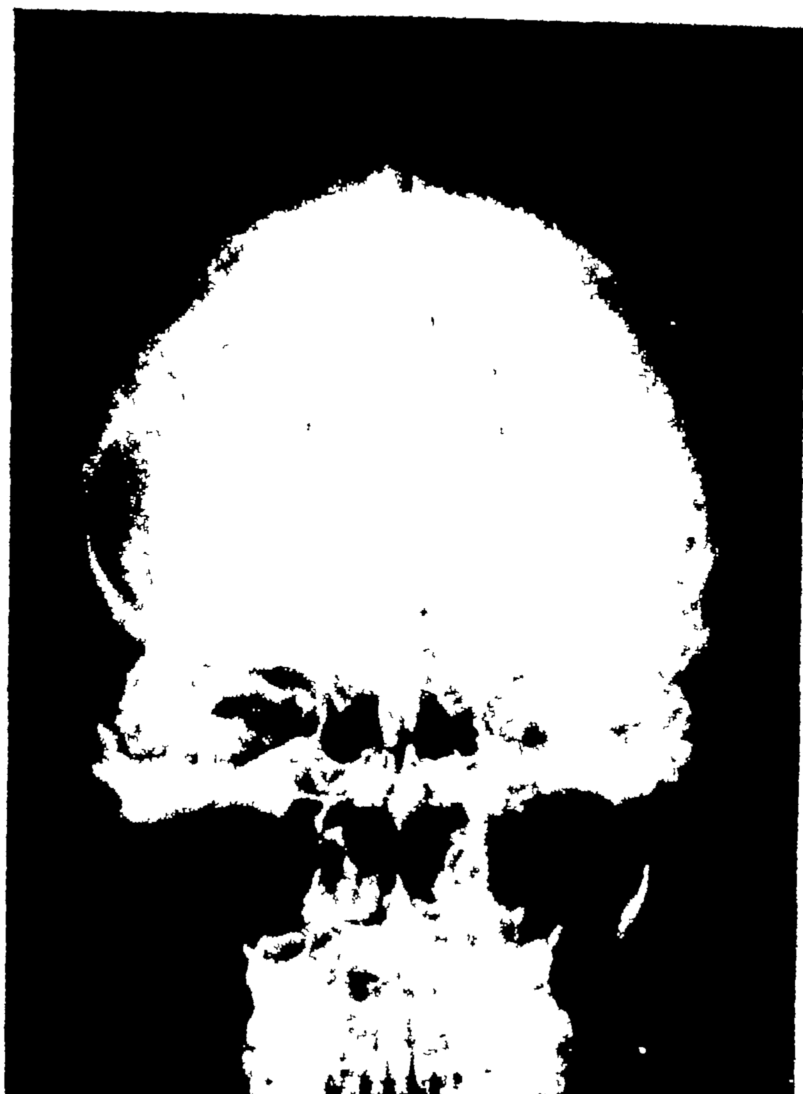


Fig 49 Fracture of the skull extending into the frontal sinus X-ray taken two months after injury

4 Frontal fractures with pneumocephalus.

Simple fractures of the frontal sinus which involve only the outer wall usually result from a direct blow to the frontal

region by a blunt object The outer wall of the sinus may be slightly depressed The symptoms depend on the degree of resultant brain contusion and laceration, which however in many cases with injuries of this type, is minimal The treatment is rest in bed in a semi sitting position Coughing sneezing straining and blowing the nose should be avoided Local treatment to the nasal passages is contraindicated After several weeks, if a depression of the outer sinus wall exists, and is objectionable for cosmetic reasons, it may be elevated under local anesthesia

Compound fractures of the frontal sinus without cerebrospinal fluid rhinorrhea or pneumocephalus require simple débridement of the scalp laceration and suture after roentgenograms have been taken If the outer sinus wall is badly depressed it must be elevated, and at the same time the entire sinus explored for fracture of the inner wall and possible laceration of the dura mater The method which Teachenor advises is adequate for these injuries The sinus wall is exposed. If the inner wall is fractured and the dura mater torn, the inner wall must be removed and the dural laceration securely sutured. A drainage tube extending through the frontal duct into the nasal cavity or externally through one corner of the wound is inserted The following case illustrates this method

Case 14 A white female 53 years old, was struck by a truck She was unconscious on admission to the hospital about thirty minutes later There was an extensive laceration in the mid frontal region extending to an underlying fracture of the skull The pulse rate was 64 and the blood pressure 124/76. The deep reflexes were all hyperactive and bilateral Babiński signs were present. Débridement was done under local anesthesia. The edges of the scalp laceration were trimmed An extensive depressed fracture involving the frontal sinus was disclosed. The depressed fragments were elevated and removed The posterior wall of the

frontal sinus was fragmented and the dura over the right frontal pole was torn The defect in the dura was repaired with a piece of frontalis fascia The mucous membrane of the right frontal

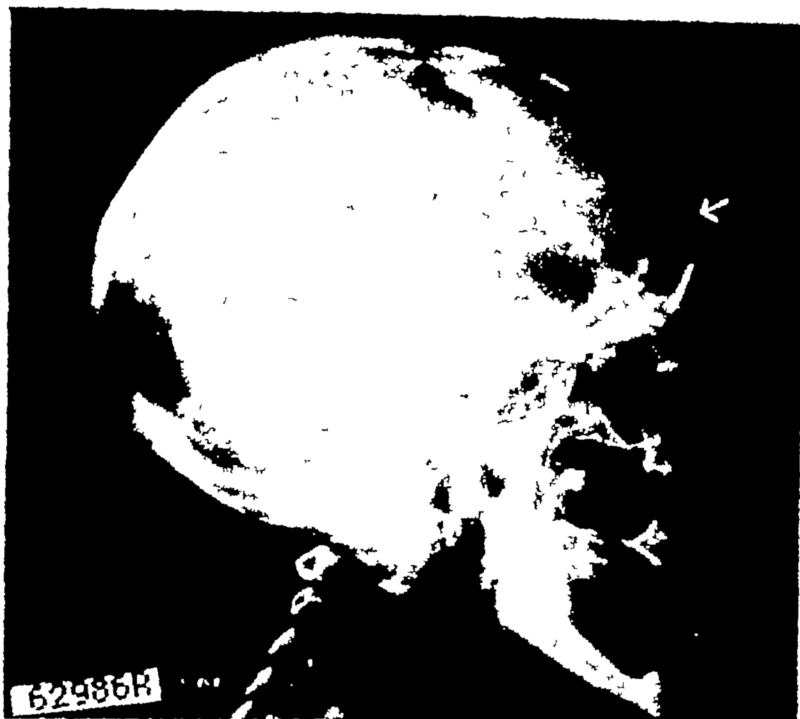


Fig 50 Case 14 Defect in the skull following repair of a fracture extending into the frontal sinus with rupture of the inner plate and herniation of the dura

sinus was removed A rubber tube was placed in the cavity of the frontal sinus for drainage The scalp edges were approximated with fine black silk, using one layer for the galea and one for the skin The drainage tube had its exit from the inner corner of the wound The patient remained in a semistupor for several days The drainage tube was removed on the third day The patient slowly improved and left the hospital six weeks after the accident. An x-ray film of the skull shows the defect in the frontal region resulting from the débridement (Fig 50).

When there is a fracture of the skull with cerebrospinal rhinorrhea, we can be certain that there is a tear in the dura mater and arachnoid membrane, and that this communi-

cates with the paranasal sinuses. The escape of cerebrospinal fluid from the nose immediately following fractures which involve the paranasal sinuses may at first be masked by the accompanying profuse nasal hemorrhage

Cerebrospinal rhinorrhea must be suspected whenever a watery discharge from the nose occurs and increases when the patient is recumbent If any doubt exists some of the fluid should be collected and tested chemically Cerebrospinal fluid is easily identified by its low protein content and the presence of reducing sugar whereas mucus has a high protein content and reducing sugar is absent The symptoms depend on the degree of the associated cerebral injury With severe contusions and laceration of the frontal lobe, shock is often profound There may be deep coma alternating with marked restlessness With extensive fracture of the cribriform plate brain tissue may escape from the nose along with the cerebrospinal fluid On the other hand some patients are in relatively good condition from the start the only symptom being the rhinorrhea, which is sufficient in itself to cause grave concern

The treatment of these cases demands the exercise of the best clinical judgment One must weigh the risk entailed by an operation against the hazard of intracranial infection in each case

The condition of the patient is the all important criterion in deciding whether conservative or operative treatment should be carried out A patient who has sustained an obviously severe intracranial injury and who is in shock or deeply comatose should not be operated upon until the shock has been treated Patients operated upon in deep coma rarely recover

On the other hand operative intervention offers the best chance of escaping an intracranial infection in young, robust

individuals who recover from the immediate effects of their injury within a few hours, but in whom cerebrospinal rhinorrhea persists for twenty-four hours or more, in spite of conservative therapy

When the fracture is directly over the frontal sinus and involves the inner sinus wall, early operation is indicated since fractures in this location can be repaired by a relatively simple procedure When it is probable that the dural tear is secondary to a fracture of the floor of the frontal fossa so that a frontal bone flap is necessary, a delay of twenty-four to thirty-six hours may be permitted while conservative therapy is being carried out If the rhinorrhea persists or increases, and the general condition of the patient is satisfactory, operative intervention is justified

In rare cases of fractures of the skull involving the paranasal sinuses, air is forced into the intracranial cavity through the dural tear with resultant traumatic pneumocephalus. The terms "intracranial pneumatocele" or "aerocele" have also been applied to this relatively rare complication Pneumocephalus is practically always associated with cerebrospinal fluid rhinorrhea at some time or other. Air is forced into the intracranial cavity when the pressure in the paranasal sinuses is raised by coughing, sneezing, straining, or blowing the nose Pneumocephalus is rarely suspected clinically, though in any case of cerebrospinal fluid rhinorrhea it should be thought of, especially if the patient complains of peculiar sensations on moving his head from side to side The x-ray examination confirms the diagnosis. Air may be found in the subdural space, in the arachnoidal sulci and cisternae, in the substance of the brain, in the ventricles, or in any combination or all of the above The symptoms of pneumocephalus are severe headache, irritability, confusion, and drowsiness. In addition, convulsions, hemiplegia, and a slow pulse may

be found Meningeal signs may be present, especially if the pneumocephalus has been present long enough to permit meningeal spread of infectious material.

Case 15 A 57 year old American sailor was injured in a shipwreck. He was unconscious for an unknown interval and received emergency treatment at a hospital where a scalp laceration in the left parietal region was sutured He was permitted to return to his home after twenty four hours He remained at home alone for five days at the end of which interval his wife returned from a trip and found the patient very drowsy vomiting and complaining of severe headache He was taken to the hospital where examination revealed a very irritable patient complaining of severe headache A thin watery fluid escaped from the left nostril when the patient was in a recumbent position The pulse was 56 Although examination was difficult there seemed to be some hesitation in naming objects (anomia) The reflexes on the right side were increased. Roentgenograms of the skull showed both lateral ventricles filled with air and a large collection of air over the left frontal lobe (Figs. 51 52) The ventricular system showed a slight displacement to the right A linear fracture of the right parietal region was present and this extended into the base of the skull in the region of the sphenoidal sinus

The next day the patient developed neck rigidity and the temperature was elevated. Lumbar puncture yielded a cloudy fluid containing 11 000 white blood cells per c. mm The patient expired several days later Postmortem examination showed a thickened pia arachnoid Part of the orbital surface as well as the first and second frontal convolutions were destroyed. There was a smoothly lined cavity 4 cm in diameter in the white matter of the left frontal lobe. In a horizontal section of the brain a communication existed between the cavity and the left lateral ventricle (Fig 53)

When this type of case is seen and diagnosed early enough a repair of the dural laceration may prevent the development of meningitis

The conservative treatment of cerebrospinal rhinorrhea when indicated is

1 The patient must be warned not to blow his nose, cough, strain or sneeze If necessary, sedatives and small doses of codeine may be given.



FIG 51 Case 15 Traumatic pneumocephalus X-ray taken with occiput on the film Note displacement of the lateral and third ventricles and collection of air in the left frontal region

2 He should be kept in a semi-sitting position in bed.

3. Local treatment to the nasal passage must be avoided.

4 Moderate dehydration to reduce the secretion of cerebrospinal fluid and aid in the sealing off of the dural tear.

5. Sulfanilamide and related drugs should be administered

when streptococcal infection is feared. These drugs are fo in high concentration in the cerebrospinal fluid after ora intravenous administration Recent investigations indic

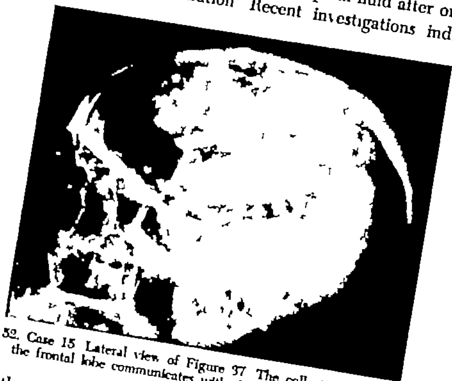


FIG. 52. Case 15 Lateral view of Figure 37 The collection of air within the frontal lobe communicates with the left lateral ventricle

that they are also efficacious in other types of meningeal in fection

Surgical intervention is indicated in fractures of the skull involving the nasal accessory sinuses whenever the risk of an intracranial infection is greater than the risk of the opera tive procedure The evalution of these factors is often diffi cult and requires astute clinical judgment When brain tissue escapes from the nose or when pneumocephalus is present, operation should be done as soon as the patient s condition permits This applies also whenever cerebrospinal fluid rhi norrhea persists for more than thirty-six hours

When the fracture involves the frontal sinus a relatively simple method often suffices This consists of exposing the

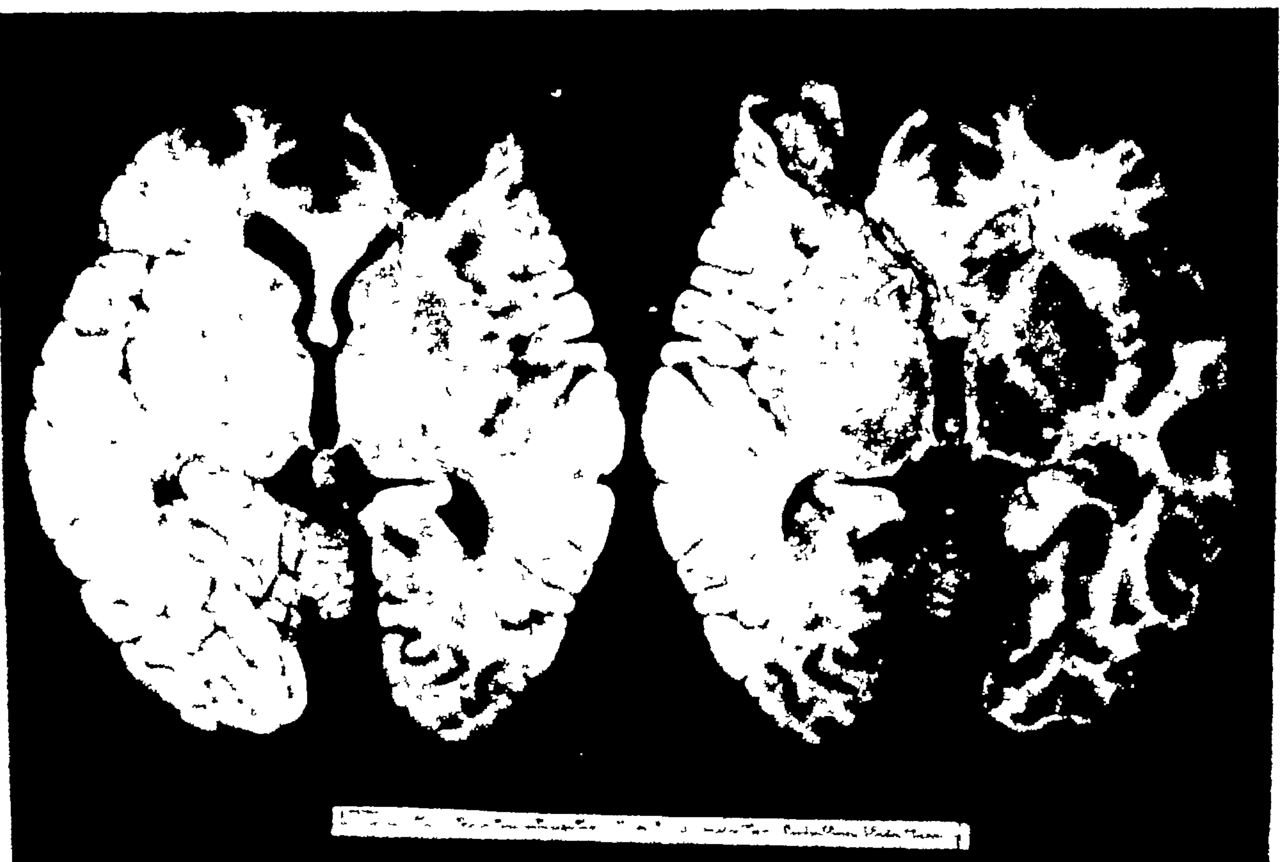


FIG. 53. Case 15. Horizontal section of the brain. Note the communication between the traumatic cavity in the left frontal lobe and the ventricular system.

outer wall of the frontal sinus by means of an incision through the shaved eyebrows. The sinus is opened with a drill, and the opening enlarged with rongeurs The clot in the sinus is removed and the inner sinus wall exposed and removed As a rule the dural tear will be in the neighborhood of the fractured inner wall Blood clot and lacerated brain are removed by suction and irrigation with Dakin s or saline solution The dural tear is then carefully repaired using a piece of fascia lata from the patient s thigh if necessary, or a piece of frontalis muscle The comminuted bone fragments are removed and discarded A small rubber tube is placed through the nasofrontal duct with its exit into the nasal cavity The inner end of the tube is placed at the mouth of the duct The drainage tube is removed at the end of about seven days At the end of this interval the dural laceration should be well healed

When rhinorrhea persists and the x ray films reveal a basal fracture in the anterior fossa or when a traumatic pneumocephalus is present a search for the laceration in the dura mater covering the floor of the anterior fossa is advisable. This is best accomplished by means of a small transfrontal bone flap (see Chapter XV) When the dura mater is separated the inner wall of the frontal sinus and the cribriform region can be inspected The dural rent may sometimes be very small If macerated brain tissue is present it is removed by suction and irrigation The tear is then repaired with fine silk sutures using a fascial transplant if necessary The transplant may be obtained from the fascia of the temporal muscle which is readily available The wound should then be closed in layers with fine silk sutures In the postoperative care of these patients in addition to the usual precautions following craniotomy they must be warned against blowing the nose,



Fig 53 Case 15 Horizontal section of the brain Note the communication between the traumatic cavity in the left frontal lobe and the ventricular system

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sneezing, straining or coughing They should be kept in bed in a semi-sitting position for at least fourteen days.

Early operation in these cases has substantially reduced the incidence of intracranial infections The expectant treatment of fractures involving the nasal accessory sinuses with persistant cerebrospinal fluid rhinorrhea or intracranial pneumatocele has little to recommend it The risk of meningitis and brain abscess is great, not only within the period immediately following the accident, but also many weeks or months later when any new infection of the nasal passages may invade structures made vulnerable by the previous injury

FRACTURE OF THE SKULL WITH INVOLVEMENT OF THE MASTOID AND MIDDLE EAR

Fractures of the middle fossa of the skull frequently involve the middle ear, mastoid, and external auditory meatus. While such injuries are usually accompanied by a bloody discharge from the ear, a discharge of blood or cerebrospinal fluid from the ear practically always means a basal skull fracture Often the aural symptoms are overlooked at first because the condition of the patient precludes careful hearing tests. When the patient recovers from shock and coma, attention is drawn to the ear symptoms

The bloody discharge from the ear may be prolonged and very profuse, especially when a branch of the middle meningeal artery or the lateral sinus is torn In most cases, however, the bleeding ceases spontaneously after a few hours

In an occasional case, slight aural bleeding may result from a traumatic rupture of the tympanic membrane without a skull fracture. Nevertheless, a good rule to follow is. Whenever bleeding from the ear occurs after a head injury, suspect

a basal skull fracture and treat it as such until proved other wise

In a small proportion of fractures of the mastoid no blood is found in the external auditory meatus but bleeding occurs into the middle ear without rupture of the drum and on examination the drum has a characteristic dull, bluish appear ance Ecchymoses over the mastoid process (Battle s sign) appearing several hours after the injury is another indica tion of a basal fracture in this region

The escape of cerebrospinal fluid from the ear occurs much less frequently than bleeding It indicates that the dura mater and the arachnoid membrane have been torn and that a communication exists between the subarachnoid space and the middle ear In the presence of pre-existing infection of the middle ear the hazard of spreading meningitis is great While cerebrospinal fluid otorrhea usually indicates a severe injury we have seen an occasional case where the otorrhea was the only grave sign or symptom which persisted after the patient recovered from the immediate effects of the injury

Case 16 A 38 year old salesman fell off a soapbox during a political rally He struck his head on the pavement and was ren dered unconscious. When brought to the hospital he was con fused and irrational and the reflexes were increased on the left side There was an escape of cerebrospinal fluid from the right ear An x ray film of the skull (Fig 54) showed a linear fracture in the posterior half of the right parietal bone extending into the petrous ridge After twenty four hours the patient was men tally clear and had no subjective complaints Hearing was di minished on the right and the cerebrospinal fluid otorrhea per sisted for eight days. The patient remained in bed for three weeks. The hearing loss persisted, and the only other sign when he was examined subsequent to discharge from the hospital was a slight unsteadiness when attempting to balance on either foot.

Impairment of hearing accompanies many cases of injury

in this region The deafness as a rule is a combination of middle ear and nerve deafness. The conduction apparatus is interfered with by blood in the middle ear, while the audi-

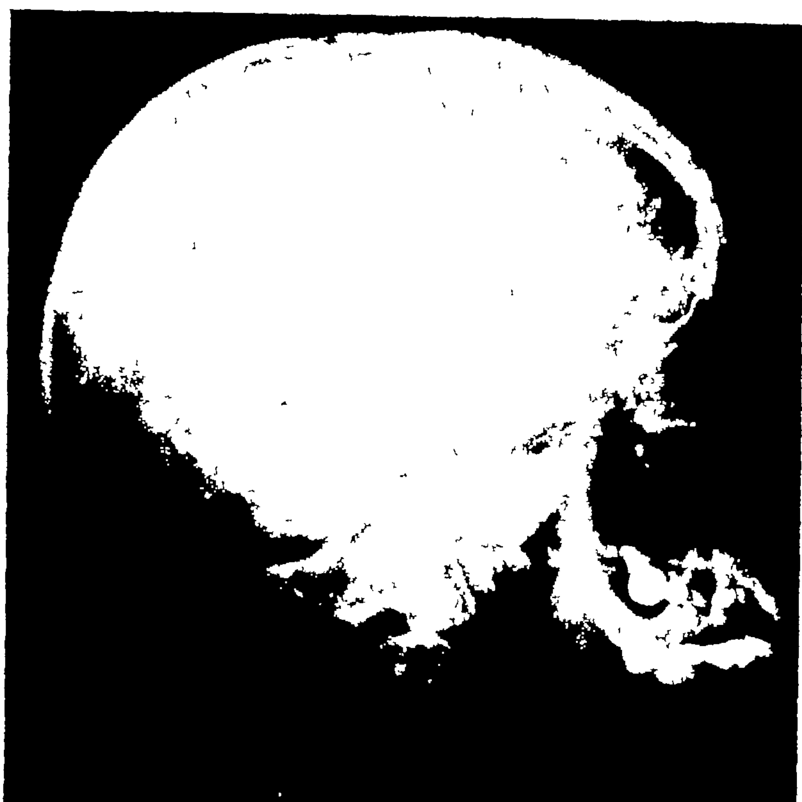


Fig 54 Case 16 X-ray film shows a linear fracture of the right parietal bone extending into the petrous ridge

tory nerve may be damaged by hemorrhage or actual laceration Patients who recover from bilateral middle fossa fractures are often left with a bilateral permanent defect in hearing

Fractures of the middle cranial fossa may extend into the labyrinthine capsule. Vertigo, tinnitus and nystagmus, accompanied by a cerebrospinal fluid otorrhea are presumptive evidence that such an extension has occurred

Conservatism is the keynote in the management of frac-

tures of the base with bloody or cerebrospinal fluid otorrhea Anything which might tend to introduce infection must be avoided. No attempt should be made to examine the ear drum or to remove the clot from the external auditory meatus by manipulation or irrigation A loose antiseptic dressing may be placed over the ear and the patient kept in bed in a semi sitting position All those measures indicated in any severe craniocerebral trauma are employed Spinal punctures are contraindicated as a rule.

The mortality in basal fractures with unilateral otorrhea is about 35 per cent, whereas when the escape of blood or cerebrospinal fluid is bilateral the mortality rate is doubled In the patients who recover impairment of hearing may persist permanently The other sequelae are similar to those following any severe head injury

The complications of basal fractures with otorrhea are

1 Peripheral facial palsy
2. Otitis media
3. Mastoiditis
4 Meningitis
5 Brain abscess

Peripheral facial palsy occurs in about 10 per cent of fractures of the base involving the petrous and mastoid portion of the temporal bone The palsy rarely comes on before the second or third day and is probably due to bleeding or edema around the facial nerve in the fallopian canal In patients who can be tested, it will be noted that taste is lost on the anterior two-thirds of the tongue on the affected side since the nerve is involved central to the branching of the chorda tympani. In most cases recovery takes place within six or eight weeks after the injury During this time mechanical splinting of the involved facial musculature aids the return of normal function During convalescence physiotherapy

consisting of massage and electrical stimulation is of value.

Purulent otitis media follows hemorrhage into the middle ear whenever the clot becomes infected. This happens rather frequently The otitis media, however, is usually benign, and indolent, but in most of the cases clears up with conservative therapy Nevertheless, in rare cases it persists and spreads, involving the mastoid, and may require operative intervention

Primary traumatic mastoiditis which does not follow otitis media is a more unusual complication of basal skull fracture. Direct trauma over the mastoid process producing a *locus minoris resistentiae* and the lodgment there of blood-borne bacteria is the probable cause The indications for surgical intervention are similar to those for mastoiditis in general The cooperation of the otologist is indispensable in all aural complications of head injuries Operative procedures on patients who are recovering from acute head injuries should be undertaken only when the indications are inescapable and then they are preferably done under local or basal anesthesia, or a combination of the two Inhalation anesthetics are to be avoided If a mastoidectomy is done, hammer and chisel must not be used, the mastoid should be opened with a drill and rongeurs, since the jarring of the skull and brain may be harmful to a patient who has had a head injury.

Meningitis following bloody and cerebrospinal fluid otorrhea (basal fractures involving the petrous and mastoid portion of the temporal bone) is particularly prone to develop if a pre-existing acute or chronic otitis media was present at the time the injury was sustained. Meningitis has followed cases of otorrhea when attempts were made to wash clots out of the external auditory meatus by irrigation. When cerebrospinal fluid otorrhea occurs in a patient with chronic otitis media, the likelihood of the development of an intracranial

extension of the infection is so great that when the patient's general condition permits an operative procedure to remove the infective material and to close the tear in the dura mater is justifiable The dural tear is best approached through a clean field by an opening in the skull in the posterior temporal region just above the external auditory meatus. This will enable the surgeon to locate the tear and close it If the tear is inaccessible for suturing it may be necessary to seal it off with a muscle graft Following this procedure, the otologist should remove the necrotic and purulent material from the mastoid through a separate mastoid incision

Brain abscess may follow traumatic otitis media and mastoiditis The pathogenesis is similar to that of nontraumatic otitic brain abscess

Case 17 A 27 year old truckman was struck on the left temporoparietal region by a heavy box He was stunned but was able to continue at work Two days later he noticed a purulent discharge from the left ear Three weeks after the accident a left mastoidectomy was performed Two weeks later the patient became dull, nuchal rigidity set in, and the pulse dropped to 58. The spinal fluid at this time was cloudy and contained 700 W B C. per c mm The next day he became hemiplegic on the right side and deeply comatose and at this time came under our observation. In addition to the above findings a bilateral papilledema was present. A diagnosis of a left temporal lobe abscess was made this was drained at operation Improvement followed for seventy two hours The state of consciousness cleared sufficiently to demonstrate a marked aphasia Motor power improved on the right side of the body However signs of meningeal infection became manifest, and progressed, terminating with the death of the patient on the sixth postoperative day

This case was under treatment before the advent of sulfanilamide. The outlook in similar cases treated with sulfanilamide and related drugs is decidedly better

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postulated and the hazard of meningitis is greater Half of the deaths from meningitis complicating head injury fall into this group Meningitis is especially prone to develop follow



FIG. 55 Photomicrograph of cerebral cortex and leptomeninges from a patient who died of meningitis following a fracture of the skull extending into the frontal sinus

ing a fracture of the petrous portion of the temporal bone if a pre-existing chronic otitis media or mastoiditis is present at the time of the head injury For these reasons the condition of the patient permitting it is advisable to attempt to repair the dural tear through a clean field, and then have the otologist perform a mastoidectomy (page 151)

Fractures of the skull which involve the paranasal sinuses

Chapter XI

SUPPURATIVE CRANIAL AND INTRACRANIAL DISEASE RESULTING FROM TRAUMA

POST-TRAUMATIC MENINGITIS

Meningitis is responsible for about 5 to 8 per cent of all deaths following head trauma In a series of 444 cases studied by Gross and Koffler there were 54 deaths Of these, 3 were due to meningitis. The hazard of meningeal infection exists in any case where the dura mater has been torn, especially if a portal of entry is opened for the passage of infective material into the subarachnoid space

In compound skull fractures, either simple or depressed, where the dura mater has been torn, the threat of meningitis exists but can be almost completely eliminated if the wound is thoroughly and promptly débrided according to the principles outlined in the chapter on the treatment of compound skull fracture (page 87) When, for some reason, such injuries are not promptly débrided a certain number of the patients develop meningitis. The symptoms of this complication usually manifest themselves within three to eight days after the injury In rare cases the infection remains latent and signs of meningitis may appear weeks or months after the patient has been discharged in apparently good health.

Basal fractures involving the petrous pyramid with bleeding from the ear furnish a portal of entry into the subarachnoid space, and meningitis may develop. In the presence of cerebrospinal fluid otorrhea, a tear in the dura mater must be

postulated and the hazard of meningitis is greater Half of the deaths from meningitis complicating head injury fall into this group Meningitis is especially prone to develop follow



FIG. 55. Photomicrograph of cerebral cortex and leptomeninges from a patient who died of meningitis following a fracture of the skull, extending into the frontal sinus.

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Fractures of the skull which involve the paranasal sinuses

especially when cerebrospinal fluid rhinorrhea is present, expose the patient to a definite threat of meningitis (Fig 55) The treatment of these fractures, which account for about 3 per cent of all deaths following head injury, has been discussed in Chapter X

The symptoms of meningitis are well known. However, in a patient with a head injury, there is often a tendency to attribute the symptoms to the intracranial damage resulting from the trauma A patient who develops high temperature, vomiting severe headaches and stiff neck following a head injury that can be included in the above categories, must be suspected of having a meningeal infection, and a spinal tap is indicated without further delay. The diagnosis of meningitis is confirmed by the presence of a turbid or cloudy cerebrospinal fluid which contains many pus cells, and in which the glucose is absent or greatly reduced Organisms can usually be found on smear and culture Meningococcal meningitis complicating fracture of the skull has been reported from time to time The factors involved are similar to those of the other meningitides complicating skull fractures except that the invading organism happens to be the meningococcus In the treatment of meningitis following head injuries the following principles are of value

1. If the portal of entry can be established, this should be attacked directly For example, in fractures through the frontal sinuses, radical operation with removal of the focus of infection is indicated.

2 The offending organism mus[illegible] identified as rapidly as possible

3 Sulfanilamide and relat[illegible] [illegible]fic antisera and antitoxins shoul[illegible] ever indicated

4 Frequent spinal punctures must be done to relieve increased intracranial pressure administer therapeutic agents and provide drainage



Fig. 56. Traumatic brain abscess This patient had a compound skull fracture two years previously He recovered from this and seemed well until he was suddenly seized with chills and high fever At autopsy the abscess was found at the site of the old injury It had communicated with the subarachnoid space producing a fulminating meningitis.

BRAIN ABSCESS FOLLOWING TRAUMA

Abscess of the brain following trauma is an uncommon complication (Figs 56 57) It may follow gunshot wounds

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2. The offending organism must be identified as rapidly as possible.

3. Sulfanilamide and related drugs, transfusions, specific antisera and antitoxins should be used without delay whenever indicated.

4 Frequent spinal punctures must be done to relieve increased intracranial pressure administer therapeutic agents and provide drainage



FIG. 56 Traumatic brain abscess. This patient had a compound skull fracture two years previously He recovered from this and seemed well until he was suddenly seized with chills and high fever At autopsy the abscess was found at the site of the old injury It had communicated with the subarachnoid space producing a fulminating meningitis.

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where infectious agents are carried directly into the brain by the missile (Chapter XII), and in some instances, compound skull fractures where the debridement has been in-

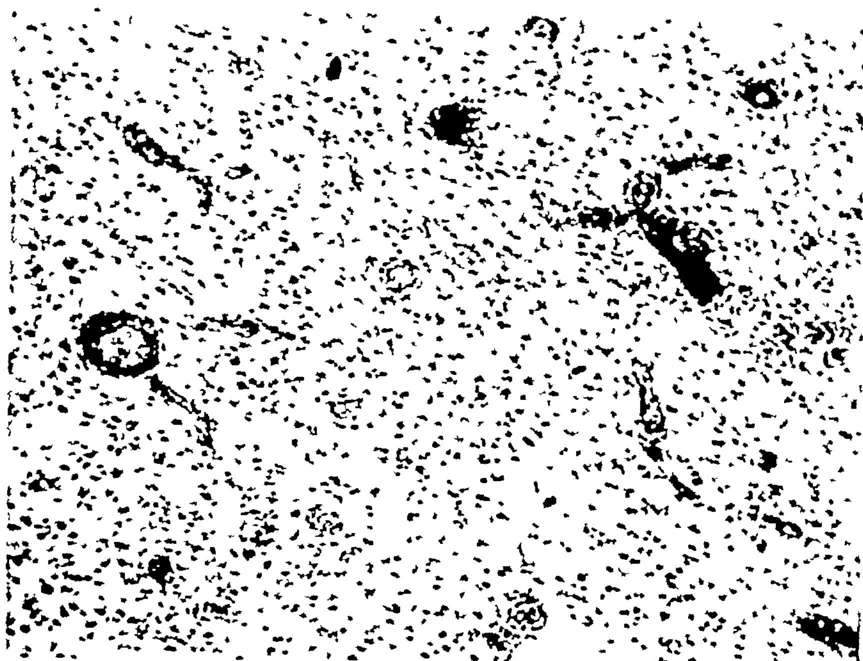


FIG 57 Photomicrograph of brain tissue adjacent to a traumatic brain abscess Note marked infiltration of the white matter and perivascular spaces with white blood cells

complete. The symptoms of abscess of the brain following trauma rarely appear until the end of ten days, and more often after an interval of two or three weeks, occasionally they may be delayed for many weeks or months After gunshot injuries, multiple cerebral abscesses may form along the track of the missile The abscess may not be found around the bullet but at any point along the track where pathogenic organisms may have been lodged In one case in which a bullet was lodged in the occipital pole, the area around the bullet was explored without disclosing an abscess Examination of the brain later revealed a large, well-encapsulated abscess in the frontal lobe near the wound of entrance Foreign bodies

may be surrounded by quiescent abscesses for many years Brain abscess complicating traumatic otitis media and mas toiditis differs in no essential character from nontraumatic otogenous brain abscess A head trauma often precipitates cerebral symptoms in a patient with a chronic or acute otitis the trauma favoring the spread of a localized infection through fracture lines extending into diseased mastoid cells and tears in the dura mater Contused and edematous brain tissue provides a suitable medium for the growth of pathogenic organisms The treatment of acute skull fractures in volving the mastoids and middle ear has been discussed in Chapter X. When an otitic brain abscess develops in a patient with a pre-existing acute or chronic ear infection, the trauma probably plays an important rôle The treatment differs in no essentials from a nontraumatic otitic cerebral abscess

CEREBRAL HERNIA

A cerebral hernia is a protrusion of cerebral tissue through a defect in the dura mater secondary to a compound fracture of the skull or some operative procedure on the skull When compound skull fractures are thoroughly and promptly débrided and a careful suture of the galea carried out, cerebral hernias rarely occur In the presence of infection however the wound may break down and if the dural closure has been incomplete or faulty brain matter will be forced through the defect in the dura mater and skull by the increasing intra cranial pressure The protruding mass may reach an alarming size (Fig 58) It consists of edematous and infiltrated brain covered by granulation tissue At the base of the cerebral hernia adhesions between the pia arachnoid and dura mater usually effectively seal off the subarachnoid space though occasionally the adhesions are not complete and cerebro spinal fluid escapes When the adhesions are firm every effort

should be made to keep them intact since meningitis is less likely to develop when the subarachnoid space is sealed off. The treatment of a cerebral hernia requires care and patience. First, the mass must be protected mechanically. This can be accomplished by the use of a very large head dressing, building up the area surrounding the hernia with cotton. The scalp surrounding the hernia for a distance of 5 cm. or more must be kept closely shaved The hernia is then covered with gauze impregnated with vaseline, or vaseline and xeroform (4 per cent) Then a large protective dressing is applied. When sloughs and necrotic areas are present the use of hypertonic saline solution (5 or 10 per cent) on the dressings is helpful Dressings of gauze soaked in azochloramid or 1:2000 acriflavine are also helpful As a rule, daily dressings are required Care must be taken to prevent injury to the hernia by bandages which are too tight. As the intracranial pressure is reduced, and as the infection subsides the hernia slowly becomes smaller and smaller, and finally covered with epithelium. At this stage, vaseline-scarlet-red gauze is helpful. When the intracranial pressure is high, daily lumbar punctures, fluid restriction, and the administration of hypertonic solutions intravenously and rectally are indicated.

During the course of treatment of a cerebral hernia the patient may have bouts of headaches, vomiting, and stiff neck, due to the spilling over of infective material or toxins into the subarachnoid space The use of sulfanilamide is valuable, if the offending organisms are susceptible to this drug

The treatment of a cerebral hernia is long drawn out, but excellent results can be obtained even after a protracted course in a seemingly hopeless case. When the hernia persists, or increases in size in spite of all the measures outlined, it is probable that a deeply situated brain abscess is present Such a diagnosis can be verified by aspiration with a long



. 58. Case 18. Large cerebral hernia re- ılting from a compound skull fracture.

Fig. 59 Case 18. Cerebral hernia.

Fig. 60 Case 18. Appearance at time of discharge

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Fig. 58 Case 18. Large cerebral hernia resulting from a compound skull fracture.



Fig. 59 Case 18. Cerebral hernia.



Fig. 60 Case 18. Appearance at time of discharge

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ion through the center of the hernia with the electrosurgical
lpel, and the introduction of a drainage tube are indicated



ig 61 Case 18 Roentgenogram of skull taken several months after
patient had been discharged from the hospital There is no evidence
iew bone formation

Case 18 A 6 year old schoolboy suffered a compound skull
cture when he ran into the side of a moving automobile. The
ient was admitted to a hospital where he became uncon-
ous and remained so for seventy-two hours. Brain and cere-
spinal fluid exuded from the open wound which was covered
h an antiseptic dressing The patient came under our observa-
eight weeks after his accident At this time there was a large

cerebral hernia in the left occipital region (Figs 58 59) In spite of dehydration and dressings of 5 per cent saline solution applied to the hernia it continued to increase in size Aspiration through the center of the hernia with a long needle yielded a large amount of yellowish green pus Accordingly a few days later the hernia was incised with an electrosurgical scalpel revealing a cigar-shaped abscess cavity This was drained by means of a rubber tube which remained in situ for five weeks, at which time the cavity had become almost obliterated. At the time of discharge the hernia was covered with healthy epithelium (Fig 60) The patient had a residual right homonymous hemianopsia due to destruction of his left occipital lobe and inability to recognize the shape of objects placed in his right hand (astereognosis) He returned to school where he learned to write and perform other skilled acts with his left hand. When seen eighteen months after the accident he was in excellent health though he still had the hemianopsia and astereognosis X ray examination of the skull showed a defect in the occipital region (Fig 61) This was well protected by scalp and hair If necessary the defect can be repaired at a later date.

TRAUMATIC OSTEOMYELITIS OF THE SKULL

Osteomyelitis of the skull following a blow to the head is not a frequent complication nowadays (Fig 62) Prior to the time of antiseptic surgery it was a common occurrence following open cranial wounds When large areas of the skull are denuded by extensive lacerations or burns of the scalp the bone is more likely to become involved Osteomyelitis may also result from compound skull fractures which become infected

The anatomical factors which influence the development of osteomyelitis are the character of the outer table the arterial supply and the venous drainage When the outer table has been bruised or fractured, in the presence of scalp laceration organisms may be introduced by the traumatic agent or from the hair follicles which are usually infected The infection

may be carried into the diploic spaces by thrombosis of emissary and diploic veins. When extensive portions of scalp are lost, leaving large areas of denuded calvarium the blood

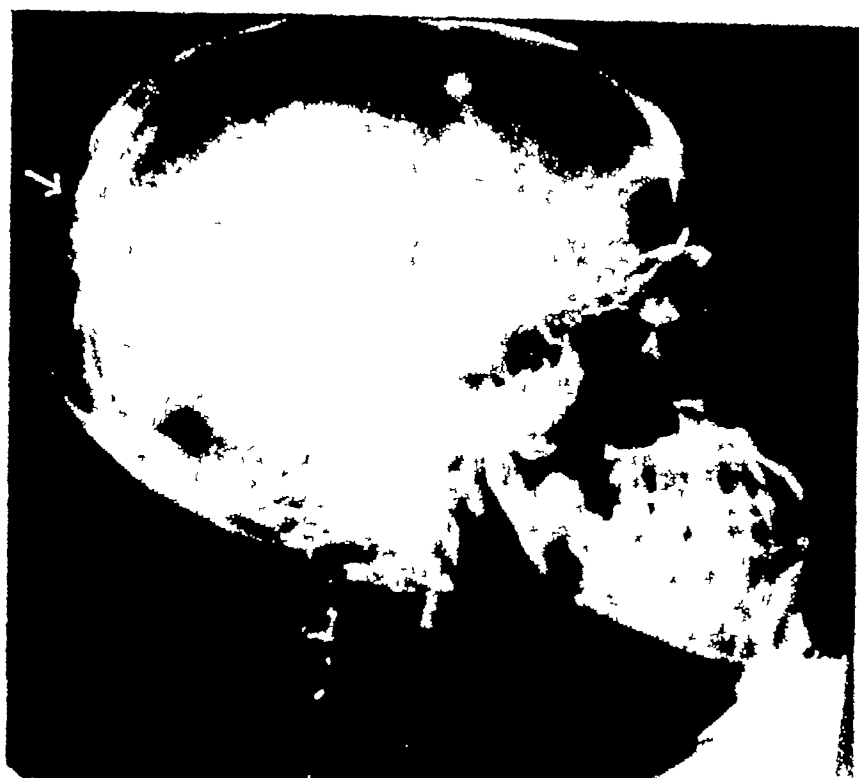


FIG 62 Traumatic osteomyelitis of the skull

supply of the outer table is impaired, since it is maintained by small arterioles from the pericranium. Degeneration of the outer table occurs and sequestra are separated. Granulation tissue covers over the denuded bone, and eventually becomes epithelialized The inner table may remain intact, since it derives its blood supply from the dura mater

The symptoms of osteomyelitis of the skull may be delayed for several days or weeks after the injury. Low grade septic temperature, localized soft doughy swelling of the scalp, or a persistent sinus following a compound skull fracture are

indicative of the presence of a suppurative process involving the bone X ray examination of the skull is usually negative in the early process of this complication and therefore of little value in the diagnosis at this stage However if repeated x ray films are taken a diffuse moth-eaten appearance can be detected after seven to ten days

Concomitant involvement of the underlying meninges and cortex by a reactive edema are indicated by various accompanying neurological phenomena such as headaches convulsive seizures neck rigidity and localized alteration in reflexes and motor power

The complications of osteomyelitis of the skull make this disease a particularly serious one. The most common complications are epidural abscess cerebral abscess meningitis and thrombophlebitis of the intracranial venous sinuses

Epidural abscess accompanies practically all cases of osteomyelitis of the skull in which both tables are involved. Cerebral abscess occurs as a result of retrograde thromboplebitis of the small veins which pass between the dura mater and cortex and may occur without concomitant thrombophlebitis of the dural sinuses The symptoms are similar to those in any case of cerebral abscess Headaches vomiting stupor bradycardia, and papilledema are of frequent occurrence Meningitis is due to a spread of the infection from the extradural space along the venous channels into the subarachnoid space

The modern treatment of diffuse osteomyelitis of the skull began when McKenzie advocated radical removal of full thickness of the skull including inner and outer table well beyond the involved areas The choice of an anesthetic depends on the condition of the patient. In general avertin supplemented by some inhalation anesthetic is preferred However when general anesthesia is contraindicated local infiltration may be employed if extreme caution is exercised

The solution should be injected well beyond the edematous area of the scalp. For cosmetic reasons, the scalp incision in outlining the flap should, if possible, be kept within the hair line. Bleeding from the scalp may be controlled by the use of artery clamps on the galea, or Michel clips, as described in Chapter XV. The scalp flap including the periosteum, is carefully stripped from the calvarium Subperiosteal abscesses are frequently encountered. Several burr holes are made to one side of the involved area in healthy bone. The entire area can then be removed *en masse* by connecting the burr holes with channels made by rongeurs Gigli saw cuts are not advisable because of the greater risk of injuring the dura mater in passing the guides. Should infected material be introduced into the subarachnoid space, the hazard of a spreading meningeal infection is increased All diseased bone must be removed The area of bone removed corresponds roughly to the area of edematous scalp A pack saturated in acriflavine 1:2000 is placed between the dura mater and scalp flap, which may then be sutured into place with a few retention sutures to prevent retraction In applying the head dressing acriflavined gauze is placed next to the scalp flap. A Dakin tube is then allowed to lead out from this area so that the adjacent dressing can be kept moist by frequent injections of acriflavine through the Dakin tube. The wound is dressed after forty-eight hours The gauze pack between the scalp and dura mater is gently loosened and irrigated with acriflavine solution. Thereafter dressings are changed daily and the gauze pack gradually removed When the wound appears clean, the scalp flap is sutured loosely in good approximation, leaving in place one or two rubber tissue drains which are gradually removed in three to five days as indicated

Chapter XII

GUNSHOT AND OTHER PENETRATING WOUNDS OF THE HEAD

GUNSHOT WOUNDS

Gunshot wounds of the head, in civil practice, result from attempted suicide or homicide, or from accidents They differ from gunshot wounds observed in military practice in that they are nearly always inflicted at close range. In military practice the ratio of gunshot wounds of the head to the total number of casualties has always been relatively high Statistics obtained from various treatises on military surgery during recent wars indicate that approximately 20 per cent of all wounds inflicted in combat are head wounds

Gunshot wounds of the head may be divided into three groups

1 Scalp wounds
2. Wounds of skull without injury to the cranial contents
3 Wounds of skull with injury to the cranial contents.

Gunshot wounds of the scalp alone are rarely serious In military practice most of these wounds are due to superficial shots, some are produced by shell fragments and shrapnel which may cause jagged lacerations with considerable loss of tissue Gunshot wounds of the scalp are treated just as any other scalp wounds If seen early that is during the first twenty four hours or so they may be closed by primary suture The hair is first carefully shaved for a distance of at least 5 cm from the limits of the laceration The wound is then thoroughly cleaned with green soap water and alcohol

and all foreign material carefully removed. Dakin's solution or a saline solution is used freely in irrigating the wound during this procedure The contused and macerated edges

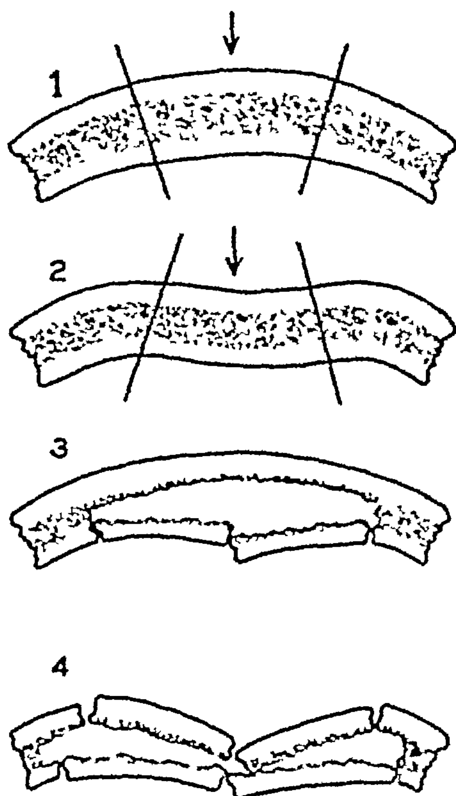


Fig 63 Mechanism of fracture of the inner table and outer table of the skull (Redrawn from "Manual of Neurosurgery," T H Weisenburg Editor, United States Army Medical Department, Washington, 1919)

are then trimmed with the scalpel. If the periosteum is contused, it, too, must be débrided. When the laceration is in the temporal or occipital region the contused fascia and muscle are treated in a similar manner. The galeal edges are then approximated with buried, interrupted, fine silk sutures, and the skin wound closed with similar suture material without drainage

The prognosis in gunshot fractures of the skull, not involving the dura mater, is good These injuries are fairly

common in military practice and are caused by tangential shots which produce a "gutter" fracture of the outer table with splintering of the inner table of the skull. Occasionally direct violence to the outer table by an oblique impact may produce a fracture of the inner table without fracturing the outer table (Figs 3, 63)

Fractures of the inner table without fractures of the outer table when a direct force is applied to the skull may be explained as follows. The force of the impact bends both tables and the intervening diploë. The particles of bone making up the outer table are crowded together while those of the inner table are dragged apart, and the latter splinters before the former. The simplest illustration of the mechanical factor involved is the often used one of the green stick broken across the knee—the side away from the applied force fractures while that nearest, bends. Fractures of the outer table alone are exceedingly rare. They have been described in war wounds in which the outer table has been grooved by the sharp angle of a shell fragment traveling at a much slower speed than a bullet.

In civil practice gunshot wounds with intact dura mater are relatively rare. Over a period of ten years only 4 cases in a series of 105 were reported by Goode from the Cincinnati General Hospital. The treatment of gunshot fractures of the skull without dural penetration is that used for any compound comminuted depressed skull fracture. This includes careful preparation and débridement of the scalp wound, the removal of all foreign materials by copious irrigation with Dakin's or saline solution and the removal of free and impacted bone fragments. If the surrounding intact bone has been contaminated with ground in foreign matter it too must be débrided with rongeurs. Gunshot fractures of the skull cause extensive splintering with many small fragments

which must be removed. However, should large fragments of bone be present, every effort should be made to preserve and replace them, in order to keep the bone defect as small as possible. Closure should be carried out as for scalp wounds, without drainage.

Tangential gunshot wounds of the vertex, which cross the midline, are frequently encountered in trench warfare. They may be accompanied by depression of the skull impinging on the superior longitudinal sinus. This results in a symptom complex, first described by Holmes and Sargent, which is dependent on interference with venous return from the cerebral veins draining the pre- and postcentral gyri. The clinical features of this syndrome are weakness and spasticity, especially marked in the distal segments of the lower extremities, and least evident in the upper extremities. The symptoms reach their maximum severity within twenty-four to forty-eight hours.

At this time there are marked tendon hyperreflexia and bilateral Babinski signs. The early rigidity and hyperreflexia are in striking contrast to the findings in acute injuries to the spinal cord, in which flaccidity with areflexia persisted for several weeks. The sensory disturbances in this syndrome are of the cortical type, the discriminatory sensibilities being most often involved. There is little or no impairment of pain and temperature sense. Evidence of marked increase in intracranial pressure appears early, with severe headache and blurring of the margins of the optic discs.

Variations in this syndrome depend on the location and degree of occlusion of the superior longitudinal sinus. When the anterior portion of the superior longitudinal sinus is involved symptoms are minimal. When the lesion involves the middle third of the sinus, motor symptoms are maximal, and

when only the posterior third is involved there may be few motor signs but marked alterations in sensation

During the World War the operative mortality in patients with symptoms indicative of occlusion of the superior longitudinal sinus was almost 40 per cent. This was in a great measure due to the fact that only the most seriously injured were operated upon The mortality in the unoperated cases was very small.

Gunshot wounds of the head with lacerations of the dura mater and without lodgement of the missile are relatively infrequent in civil practice, because most gunshot injuries are inflicted at close range In military practice they occur more frequently and accounted for about one-fifth of all cases in a series reported by Cushing The dural injury is produced either by depressed fragments or by detached indriven pieces of bone The focal symptoms depend on the area of the brain injured General symptoms as in other injuries depend on the severity of the trauma the amount of hemorrhage, and the degree of cerebral contusion and laceration After careful neurological and x ray examinations prompt and thorough débridement of the scalp bone, and damaged brain with removal of indriven fragments of bone and suture of the dura mater and scalp in layers without drainage is indicated

The prognosis with early débridement is excellent especially if treatment is rendered within the first twelve hours When operation is delayed for two or three days as often occurs under war conditions, the mortality is about 20 per cent

The most serious gunshot wounds of the head are those in which the dura mater and cortex are penetrated and the missile lodged in the brain or in which the bullet has perforated the skull—the "through and-through" wounds Unfortunately

they account for about 80 per cent of all gunshot wounds of the head in civil practice, and about 85 per cent of all war wounds in which the wounded are alive long enough to reach the casualty clearing stations.

In civil practice many *of the penetrating and perforating* gunshot wounds are suicidal, and in most cases the temporal region is involved. These patients are usually admitted to the hospital within the first hour of the injury. When seen they are nearly always in shock, deeply unconscious, and in the "through-and-through" wounds, often bleeding profusely from the sites of entrance and exit. In war practice many patients with this type of injury never reach the casualty clearing stations, since most of them are killed outright or death supervenes on the field, before there has been time to remove the wounded

Occasionally, a penetrating or perforating gunshot wound occurs without loss of consciousness and without shock. Cases have occurred in which a bullet has been found during a routine x-ray examination of the skull for supposed idiopathic epilepsy, or some other neurological disorder, only the most searching inquiries revealed an accident, many years before, in which the bullet wa[illegible] the time merely to have

in falling he had lacerated his scalp Since the wound had just been attended to by a physician who saw the patient before sending him to the hospital, the dressing was not disturbed. The



Fig. 64 Case 19 Bullet lodged in the right supramarginal region.

presence of a small amount of blood in the cerebrospinal fluid at lumbar puncture seemed to confirm the diagnosis of cerebral hemorrhage After a few days the patient improved. He regained some power in the paretic extremities and his mental condition was said by his daughter to be normal. Three weeks later an x ray film of the skull was taken This showed a bullet in the right parietal region (Figs 64 and 65) Careful examination of the wound now showed a round scar about 7 mm. in diameter the wound of entrance of a bullet which must have been fired from some distance since there were no powder marks. The patient had no enemies. The only explanation was that he had been hit by a stray bullet. The patient seemed to suffer no ill effects except an inability to recognize objects placed in the left hand and

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The following patient recently came under our observation

Case 19 A carpenter, aged 59 years, was brought to the hospital by ambulance The only history obtained was that he suddenly became dizzy while working on a flat roof, and fell Examination showed a small collodion dressing on the scalp in the right parietal region The patient was semi-stuporous When urged he would answer questions He had a left-hemiparesis. The blood pressure was 180/110, the pulse rate 84 The provisional diagnosis was "cerebral vascular accident," probably the result of arteriosclerosis and hypertension It was thought that

in falling he had lacerated his scalp Since the wound had just been attended to by a physician who saw the patient before sending him to the hospital the dressing was not disturbed The



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loss of position sense in the left upper and lower extremities He refused to consent to an operation for the removal of the foreign body

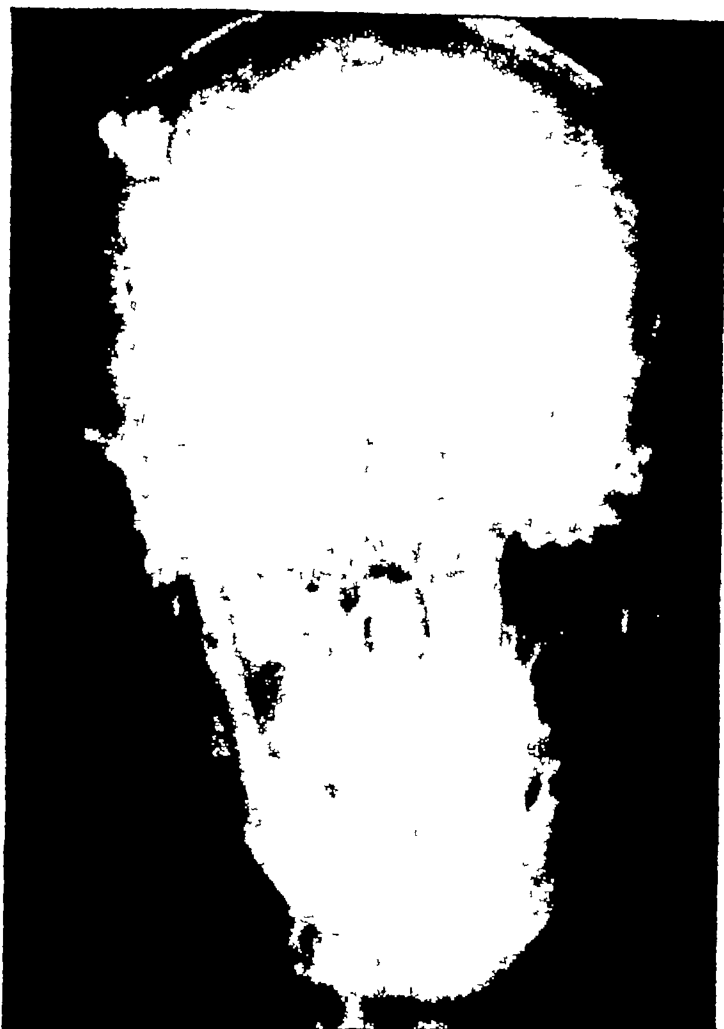


Fig 65 Anteroposterior view of Figure 64

Those few who survive "through-and-through" bullet wounds often suffer surprisingly few disabilities, due to the sparing of important structures The steel jacketed bullets with their high velocity, employed in modern warfare, have increased the number of perforating cranial wounds In

civil practice nearly all perforating gunshot cranial injuries are fatal All of the 34 patients reported by Goode died within a few hours The outstanding factor in all cases is profound shock which persists in spite of all therapy until death

In his war experience, Cushing found that when one or both ventricles of the brain were penetrated by missiles, the mortality was 100 per cent However when the bullet penetrates only one hemisphere and avoids the ventricle, as happens when the wounds of entrance and exit are on the same side and in the upper portion of the skull the outlook is somewhat better

In the management of penetrating and perforating gunshot cranial wounds the primary consideration is the treatment of shock. External heat, caffeine, 10 per cent glucose by vein and above all blood transfusion are invaluable

In civil practice nearly all severe gunshot head wounds are seen very early The question of immediate or delayed débridement must be settled Patients operated upon while in shock rarely recover, yet occasionally because of profuse hemorrhage, operation must be undertaken while shock is present In these cases shock must be treated while the operation is going on The cooperation of two operating teams is essential so that a transfusion can be given while the cranial damage is being repaired

In patients with penetrating and perforating wounds who recover from shock, the optimum time for operation is within the first twelve hours, since delay permits infection to set in in the traumatized tissues and to spread to adjacent areas. Nevertheless, operations should not be done until neurological and x ray examinations have been completed In war practice, early operation is often impossible Those who advocate delay to permit the formation of meningocerebral adhesions have been proved wrong in practice, since the

incidence of meningitis and encephalitis is greater the longer the operation is deferred

When operation within the first two or three days is not possible, expectant treatment should be carried out. the entire scalp shaved, superficial debris removed, and antiseptic dressings applied, without suture of the wounds Secondary operations are then performed as indicated

The following principles, most of them advocated by Cushing as a result of his war experience, serve as an excellent guide for the early treatment of gunshot wounds of the head.

1 Careful preparation of the field of operation. The entire scalp should be clipped and shaved, an electric hair clipper saves much time Then green soap, sterile water, alcohol, and ether should be applied for cleansing.

2 The use of local anesthesia Inhalation anesthetics are strongly contraindicated If the patient is so uncooperative that a local anesthetic is inadvisable, a preliminary basal anesthetic, such as avertin, may be employed Preliminary narcotics are contraindicated because of their depressing effect on the respiratory center However, other sedatives, such as nembutal or amytal, are of value

3 A flap or tripod incision (Fig 66) provides the best exposure The latter permits an easier closure, since by undermining the three flaps, approximation is possible even after the traumatized edges of the wound have been thoroughly débrided

4 The area of cranial perforation or penetration should be enlarged with rongeurs, or, if it is evident from the clinical or x-ray findings that a wide exploration of the brain is necessary, a bone flap, with the area of penetration in its center, should be made This is accomplished by making four or five trephine openings in the skull and connecting them with the

Gigli saw (Chapter XV) The traumatized bone in the center of the flap can then be removed with rongeurs

5 The foreign bodies and indriven fragments of bone

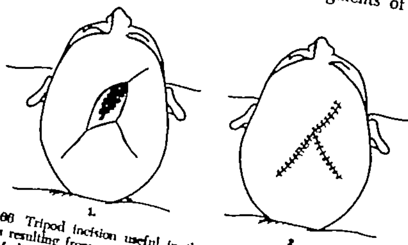


FIG. 66 Tripod incision useful in the débridement of compound skull fractures resulting from gunshot wounds. (Redrawn from Harvey Cushing British Medical Journal 1 221 1918.)

having been localized by x ray examination should next be removed. The track of the missile should be cleansed of macerated brain tissue and clot by passing a soft catheter along it and irrigating with saline or Dakin s solution The removal of metallic foreign bodies from the brain is not always possible nor indeed indicated Only when the removal can be accomplished without irremediable damage is it to be attempted During the war Cushing made use of the electromagnet applied to the magnetic foreign body by means of a rounded long wire nail passed down the track. Non metallic missiles are removed with extracting forceps The dura mater should be closed and the bone flap replaced (if one has been made) The scalp is sutured in layers—one for the galea and one for the skin with interrupted sutures of fine silk. In cases operated upon during the first twelve or twenty four hours drainage is not to be used In delayed

operations, where pus is present, or where there is brisk or continuous oozing, drainage is required.

Postoperative care demands careful and skillful nursing and observation As soon as the patient has recovered from the immediate effects of the operation he should be kept in a semi-sitting position since this tends to reduce venous pressure and thereby intracranial pressure Narcotics are to be avoided. For restlessness, phenobarbital, paraldehyde, bromides, and chloral are the drugs of choice Fluids should be maintained at about 1500 c c a day, and after the second day the bowels should be kept open, using magnesium sulphate by mouth

If headache, stiff neck, delirium, or high fever persist, a careful lumbar puncture should be done, and the fluid removed slowly The cerebrospinal fluid should be cultured If organisms are found, they must be identified as rapidly as possible and the appropriate therapy instituted The wound should be dressed after forty-eight hours and most of the sutures removed A dressing impregnated with an oily solution of azochloramid is preferred by the authors.

Hernia cerebri develops in many cases where it is impossible to close the dura mater, or where infective material remains If the hernia is the result of cerebral edema and increased intracranial pressure, the outlook on the whole is fairly favorable Extreme care must be taken in dressing the hernia The scalp must be kept shaved, and vaselined or xeroformed gauze dressings applied Lumbar punctures will benefit hernias which are due to cerebral edema, while those which result from the pressure of a brain abscess will not recede until the abscess has been drained If the hernia has an unhealthy appearance, dressings kept moist with 5 per cent saline solution will help stimulate healthy granulations and cause the sloughs to disappear (See Chapter XI.)

Meningitis brain abscess, and epilepsy are the most common complications of gunshot wounds of the head and these are discussed in their separate chapters



FIG. 67 Umbrella stay entering brain through the optic foramen.

PENETRATING WOUNDS OTHER THAN BULLET WOUNDS

Puncture wounds of the brain are the result of accidents or assaults The foreign body may penetrate the skull, usually in the thin temporal region or enter the brain through one of the foramina Foss reported the case of a child who fell while running with an ivory crochet hook in his mouth The hook traversed the wall of the pharynx and entered the left cerebellar hemisphere through the jugular foramen Figure 67 is the x ray film of a young girl who was brought to the hospital in coma, with part of an umbrella stay projecting from her orbit While at play another child thrust the umbrella

stay at her It entered the cranial cavity through the optic foramen without damage to the eye After removal of the foreign body the child recovered, but had loss of vision on the side of the injury, the result of damage to the optic nerve. Knife blades have been found in the brain years after a brawl.

In some puncture wounds of the brain there are no cerebral symptoms during life As a rule, however, signs of the initial severe cerebral injury are followed by an infection which usually terminates fatally If part of the penetrating foreign body projects above the skull it should be removed, after shaving and careful preparation of the scalp If the foreign body is buried in the brain in an accessible location and the patient comes under observation during the first twenty-four hours, it should be removed at once provided no shock exists The technique for the removal is similar to that employed in other cranial operations. If the patient comes under observation after twenty-four hours, removal should not be attempted for several weeks in order to permit localization of any infection to take place In some cases an asymptomatic imbedded foreign body is best left alone.

Chapter XIII

CRANIAL NERVE INJURIES

The cranial nerves are frequently involved in head injuries, particularly in cases of fracture of the base of the skull The lesion may be a contusion of the nerve or a hemorrhage into the nerve sheath In these cases the impairment of function is usually temporary On the other hand, when the nerve is lacerated at or near its foramen of exit from the skull, permanent loss of function ensues

The olfactory nerve filaments passing through the numerous small apertures in the cribriform plate are easily torn in anterior cranial fossa fractures The olfactory bulbs and tracts are also liable to injury due to their exposed anatomical location The defect in the sense of smell is rarely recognized during the acute phase of the head injury Inability to detect odors is usually not noticed until the patient is up and about, performing his customary duties Unfortunately testing of the sense of smell is often either neglected or impossible because of lack of cooperation so that available statistics are not accurate In reports of cases of head injuries in which special attention has been paid to the status of the sense of smell, almost complete anosmia was found in approximately 10 per cent of the cases. The loss of smell is usually permanent

Loss of vision either unilateral or bilateral, fortunately a rare occurrence is found in less than 1 per cent of head injuries. The loss of vision is due to injury of the optic nerve, chiasm or tract If the injury occurs in the nerve blindness

on the same side results If at the chiasm, bilateral blindness usually develops, while if the injury is to the optic tract, the patient will suffer from a homonymous hemianopsia Optic nerve injuries follow fractures of the frontal fossa, which extend into the optic foramen and canal In these cases loss of vision occurs at once If the nerve is severed, primary optic atrophy is apparent in from one to three weeks. When the nerve is contused and hemorrhage occurs into the sheath, the resultant venous stasis produces choked discs At first the visual loss may be complete, but as the hemorrhage absorbs there may be some return of function Decompression of the optic canal has been advocated in these cases. However, the results have been far from encouraging, since at the time the procedure would be of benefit, the patient is in no condition for such an extensive operation. Injuries to the optic chiasm and tract are relatively rare but when they do occur usually produce complete and permanent blindness.

Palsy of the oculomotor nerve is a frequent finding after head injury Its most usual manifestation is a unilaterally dilated pupil The third nerve supplies the constrictor muscle of the pupil The loss of function in this nerve may be due either to direct injury or, more commonly, to pressure of an extra- or subdural hematoma This results in dilatation of the pupil due to the unopposed action of the dilator pupillae supplied by the sympathetic nerves With greater involvement of the third nerve, ptosis, or even outward and downward rotation of the eyeball, may occur. The reason for this is the unopposed action of the superior oblique and external rectus muscles, which are supplied by the fourth and sixth nerves, respectively

Involvement of the trochlear nerve is uncommon Minor degrees of palsy due to injuries to this nerve are usually unrecognized. Injuries to the abducens nerve are more common,

having been found in 7 of 444 cases This nerve, because of its long course along the base of the skull is often injured with a resultant partial or complete paralysis of the lateral rectus muscle which is the only one it supplies In nearly all such cases, improvement in muscular strength occurred over a period of several months

The motor division of the trigeminal nerve is rarely in volved in head injuries Neither are the Gasserian ganglion nor the three main sensory divisions often damaged How ever the supra-orbital and infra-orbital nerves may be con tused or lacerated in injuries involving the orbits and superior maxilla. Marked tenderness over the foramen of exit and numbness in the distribution of the involved nerve are the sequelae

Peripheral facial palsy was found in 5 of 444 cases The facial nerve may be involved along with the auditory nerve in fractures through the petrous portion of the temporal bone Facial weakness is rarely noted immediately after the head injury, but develops after a lapse of several hours In one patient, facial paralysis developed on the fifth day It is safe to assume therefore that the injury to the facial nerve is due to hemorrhage into the nerve sheath or to edema of the nerve itself The paralysis, which is rarely complete, involves all the branches of the facial nerve If the nerve is involved above the point where the chorda tympani leaves the facial nerve (about 7 mm above the stylomastoid foramen) there will be loss of taste in the anterior two-thirds of the tongue on the affected side In most of these cases function begins to return after a few weeks and may be almost completely restored at the end of two or three months. If there is prolonged delay in recovery or if electrical tests show a complete reaction of degeneration, the outlook for eventual recovery is not good. A facial tic may develop during the period of nerve regenera

tion Most traumatic facial palsies recover spontaneously However, while waiting for recovery it is good practice to support the drooping corner of the mouth with a Y-shaped



FIG 68 Symmetrical ring fracture of the base of the skull

adhesive strip This can be anchored to the head by means of a metal band, or, in women, to the hair by means of a heavy rubber band. Electrical stimulation of the involved facial muscles may be started as soon as the patient's condition permits, and continued until function has been restored.

The eighth cranial nerve is frequently injured in head traumata, particularly when there is an associated fracture

through the petrous portion of the temporal bone Frequently the facial nerve is injured together with the auditory but the latter may be injured without any involvement of the facial nerve

When the eighth nerve is injured associated with the hearing loss one finds evidences of vestibular nerve dysfunction such as spontaneous nystagmus incoordination, tinnitus and vertigo Dizziness which is so frequently present following head injury is in most instances probably due to cerebral vasomotor disturbances Nevertheless, tests of vestibular function reveal various degrees of alteration from the normal in a high proportion of cases The degree of hearing loss varies from complete deafness to merely lessened acuity for high tones. In 444 records analyzed there were 17 cases of marked unilateral deafness and one of bilateral deafness In the latter there was a symmetrical "ring fracture" of the base extending into the foramen magnum (Fig 68)

Injuries to the remaining cranial nerves the ninth tenth, eleventh and twelfth, are usually discovered at autopsy since the associated damage to vital medullary centers is ordinarily incompatible with life

Chapter XIV

CONVULSIVE SEIZURES FOLLOWING HEAD INJURY

Convulsive seizures frequently follow an injury to the brain. They may occur immediately after the trauma, or many years later. Approximately 3 per cent of patients with major head injuries develop convulsions within the first few hours after the trauma. Seizures are especially prone to develop in the presence of a depressed fracture of the skull in or near the motor area. However, convulsions may occur with fractures in any part of the skull, and often when no fracture has been sustained. It is difficult to determine which patients will develop convulsions after a head injury. Probably other factors in addition to the actual trauma to the brain itself, are involved. The basic status of the patient plays an important rôle: those who have what may be called a "convulsive diathesis" are more likely to develop seizures, the mechanical injury to the brain being the precipitating factor. In many instances convulsive seizures follow a head injury after an interval of many months or even many years. It is difficult to estimate the percentage of patients in this group with the same degree of accuracy that is possible in a large series of patients who have convulsive seizures immediately following a head injury, because in the latter instance the seizures occur while the patient is under observation in the hospital, or shortly before he is brought to the hospital. When, however, seizures develop a considerable length of time after the injury, contact with the medical agency first treating the patient

frequently has been lost. The estimates therefore, are unreliable percentages reported have varied from 10 to 30 per cent The former figure is probably a fairer estimate and is the one generally accepted

MODE OF PRODUCTION

Convulsions are caused by an injury to the brain (Fig 69) In those cases in which convulsions come on immediately after the injury, it is assumed that the cause is laceration of the brain or subpial or intracerebral petechial hemorrhages Where the convulsions appear later they are produced by the reparative changes which occur in the brain following damage to it by trauma. In the reparative process connective tissue blood vessels and glial elements all play a rôle. Microglia and histiocytes phagocytose the destroyed elements The astrocytes (Fig 70) then proliferate Blood vessels become numerous in the injured area, and proliferation of connective tissue follows thus forming a scar composed of glial and vascular elements, which fills the defect caused by the trauma Later the scar contracts and since in so many of these cases the injured portion of the brain is near the surface of the cortex the connective-tissue elements in the scar form adhesions between the cortex and the overlying meninges (Fig 71) When the scar contracts the brain is *pulled toward the point in the meninges where the attachment has taken place* This point may thereafter be a focal point from which discharges may be set off precipitating convulsions Frequently the location of the scar may be suspected from the character of the seizures which may show some evidence of a definite aura, or may indicate the focal origin by the initial movements with which the convulsions start Not infrequently such focal points are located directly beneath an old fracture

In many cases the head injury may cause such diffuse damage to the brain, by widespread petechial hemorrhages surrounded with areas of softening, that reparative processes do



FIG 69 Cerebral scar following a gunshot wound of the head This patient was symptom-free until five years after the injury, when he began having generalized convulsions He died of pneumonia Note in addition to the scar, the agonal subarachnoid hemorrhage

not form adhesions to the overlying meninges Instead, with the contraction of the diffuse scarring, the entire brain may become atrophic. In cases of this type there is usually no localizing character to the seizures

When there is clinical evidence of the existence of a focal point, the character of the seizures depends upon the location of the scarring The scars most frequently causing Jacksonian motor seizures are those found in or near the motor cortex, and in these cases the seizures are instituted by movements of that part of the body most closely represented in the cortex adjacent to the scar Frequently, the seizures are limited to the movements of that part of the body, but in most cases the

wave of excitation spreads out resulting in a generalized seizure Sensory Jacksonian seizures are not as common as the motor seizures in the post-traumatic group of convulsions

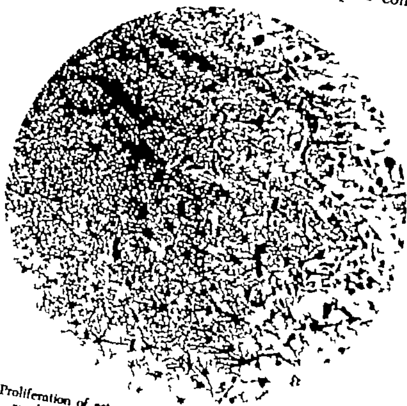


FIG 70 Proliferation of astrocytes in a traumatic lesion of the brain which resulted in convulsions. Cajal's gold sublimate stain.

Scars far from the motor area may provoke seizures which give no hint of localization since the wave of excitation by the time it reaches the motor cortex, may have spread out so diffusely that the entire body convulses at one time Scars in the prefrontal adversive area may cause seizures which begin with head and eyes turning to the opposite side and then promptly develop into a generalized convulsion Occasionally patients experience flashes of light immediately preceding a generalized seizure a symptom which casts

suspicion on the occipital region. Olfactory hallucinations preceding such convulsions bring the temporal lobe under suspicion

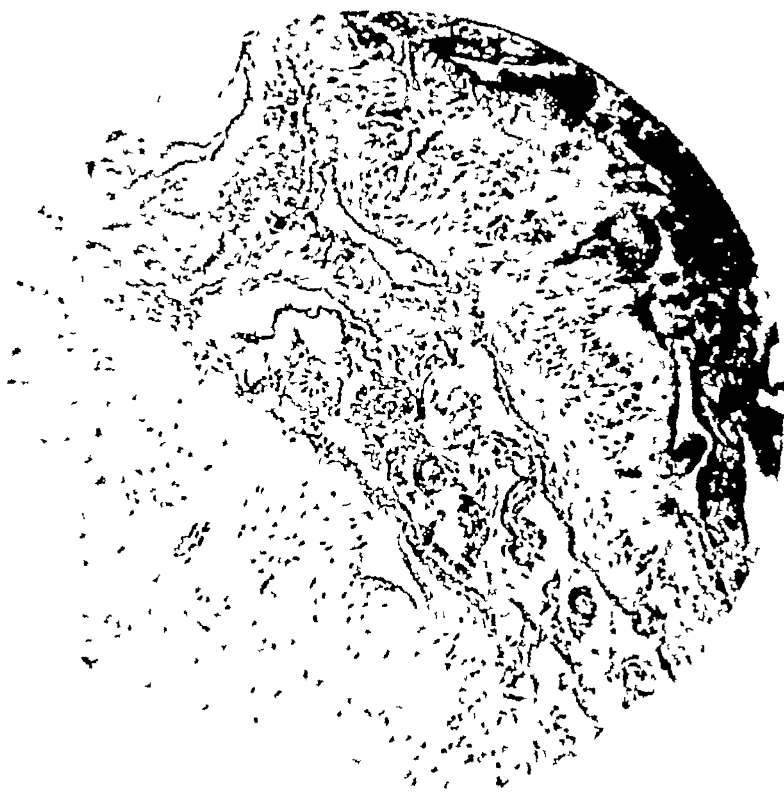


FIG 71 Cerebral scar following a compound skull fracture The adhesions between the cortex and overlying thickened dura are made up of connective-tissue elements and blood vessels

Such clinical suspicions should always be corroborated by means of air injection. This procedure may show a dilatation or prolongation of the ventricle toward the point of adhesion of the cortex to the overlying meninges, or may show a shift of the entire ventricular system toward the scar (wandering ventricle) In some cases evidence of corticomeningeal adhesions is manifested by the absence of air in the subarachnoid space overlying the involved cortex. In cases of diffuse

brain damage the shrinking of the scars results in actual contraction of the entire brain with an increase in the size of the ventricle and the subarachnoid space While at the present time electro-encephalography is of no great aid in the localization of focal points in post traumatic convulsions it offers great promise With further development in the knowledge of the rhythm of the electrical potentials emanating from the cortex and its alteration by pathological processes it is conceivable that focal points may be accurately detected by a very simple process which subjects the patient to no discomfort

TREATMENT

The most valuable and most important treatment is that instituted immediately following the head injury Depressed fractures should be elevated at the earliest possible moment Shock when present should be treated first but as soon as the patient recovers the depressed fracture should be elevated Frequently, depressions can be palpated. However it must be remembered that a subaponeurotic hematoma may confuse the diagnosis In palpating such a hematoma the periphery is frequently firm and the center soft, so that one gains the impression that a depressed fragment of bone lies beneath the center of the hematoma X ray examination of the skull should therefore, be a routine procedure whenever a depressed fracture is a remote possibility When done carefully in patients who are not in shock, there is no added risk Even when a depressed fragment can definitely be palpated clinically or actually seen through a laceration of the scalp operation should be preceded by roentgenographic studies in order to give the surgeon knowledge of the extent and type of fracture Rarely there may be multiple depressed fractures, especially with multiple injuries

Compound comminuted skull fractures should be thoroughly débrided This includes besides the scalp and bone, the underlying meninges and cerebrum. It has been shown that the early removal of traumatized necrotic tissue, leaving clean normal brain surfaces, markedly decreases the amount of scarring, and therefore diminishes the incidence of post-traumatic convulsions

Immediate conservative treatment is important as a prophylaxis In all cases of major head injury, absolute rest in bed for at least three weeks is indicated The importance of rest lies in the removal of undue strain upon the cerebral vessels whose functions have been disordered by the trauma, and characterized by the marked vasodilatation secondary to arteriolar trauma The stasis due to this paralysis of the vascular bed causes some tissue anoxemia, and as long as the vasoparalysis exists the perivascular areas of softening progress Absolute rest in bed aids the restoration of the normal vascular function, and in some cases the vasoparalysis may completely recede without great brain damage

Sedatives should be used routinely after the patient has regained full consciousness, and should be maintained regularly for a long period, depending upon the severity of the original trauma Any patient who has been unconscious for more than half an hour following a head injury should receive phenobarbital for at least six months.

Treatment, if the convulsions have already set in, is directed toward removing any existing focal point When clinical signs point to a single focal area, and corroboration is obtained by encephalographic studies, surgical attack upon that area is indicated The usual osteoplastic flap is turned down over the suspected area (Fig 72) The turning back of the bone flap must be done with extreme caution because of frequent adhesions between the dura mater and the bone

With the dura mater exposed inspection and palpation may reveal the presence of dense corticodural adhesions The dura mater should be carefully opened around this area and the



Fig. 72. Transfrontal craniotomy scar This patient suffered a compound skull fracture in early childhood. A scar just above the root of the nose remained This was attached to the dura and cerebral cortex through a defect in the skull. The first convulsion occurred about twenty years after the original accident A frontal bone flap was turned down and the meningocortical scar excised. The defect in the dura was repaired with cellophane A button of bone obtained in turning the flap was used to repair the skull defect over the cerebral scar The patient remained free of convulsions

adhesions carefully separated If a brain scar presents on the surface it is excised widely and completely Frequently the

core of the scar extends down to the ventricle, and in these cases, after the removal of the scar, free communication should exist between the ventricle and subarachnoid space. All scarred areas are removed, leaving a clean fresh area of normal brain tissue. If the dura mater is markedly involved by the adhesive processes it should be removed. The value of replacing the dura mater with fascial transplants or foreign bodies (cellophane or cargyle membrane), is questionable. Leaving the dural defect as it is, and merely replacing the bone flap seems to be equally, if not more, effective.

Occasionally nothing more than corticodural adhesions are found. These should be carefully separated. In some cases, on reflecting the bone and dural flaps no gross abnormalities of the cortex are visible. Careful electrical stimulation of the cortex may be helpful in producing attacks which simulate in all respects the clinical convulsions. If a localized area is found, in which stimulation consistently and repeatedly produces such an attack, and in which the surrounding areas do not react similarly, it should be excised, preferably by a subpial resection.

On turning back the flap the surgeon may occasionally see a localized area of yellow degeneration involving a small portion of one gyrus. Such an area should be excised by subpial resection, particularly when suspicion points toward this as a focal point.

Case 20. A male clerk, 25 years of age, was injured when, while repairing a tire in the road, he was struck by a passing automobile. He was taken to a hospital where he was found to have a left hemiparesis. He was unconscious for several days. After a prolonged hospital stay he was discharged, still hemiplegic. At home he began having tonic and clonic seizures beginning in the toes of the left foot and spreading to the left leg, thigh, and through the left upper extremity. Occasionally the seizures became generalized. They increased in frequency and severity and

were not influenced by sedative drugs. He came under our care about eighteen months after the accident. At this time he was having several seizures daily. Between attacks he had a "cramp-like" feeling in the toes of the left foot. The left lower extremity was powerless, and the left upper extremity practically so. The reflexes on the left side were increased with a left ankle clonus and a left Babinski sign. X-ray examination of the skull was normal. Encephalogram showed a slight dilatation of the right lateral ventricle.

Because of the focal nature of the convulsions, an exploration was done under local anesthesia through a right parietal osteoplastic bone flap, which extended to the midline. When the dural flap was made, adhesions between the dura mater and the cortex were found, corresponding to the motor foot area. When the adhesions were separated an area of degenerated cortex about 1 x 1.5 cm. was disclosed. This was deep yellow in color and easily removed. The patient made an uneventful recovery. He had three seizures on the first postoperative day, but none since (two years). Power returned rapidly in the left upper extremity, thigh and leg, proving that the weakness before operation was post-convulsive. There was no return of motion in the left toes or ankle, and this was to be expected from the operative findings. Even though sedative drugs had no influence on the seizures before operation, they were administered postoperatively for several months, as they should following every craniotomy for post-traumatic seizures.

There is no justification for the indiscriminate excision of a wide block of normal appearing brain in order to effect a communication between the ventricle and subarachnoid space. Valuable brain function may be interfered with by such a useless procedure, which should be limited to those cases in which the scarring is so extensive that it actually extends from the cortex down to the ventricle. Following operation the patients should be kept on phenobarbital or other anti-convulsive therapy for at least two years.

PROGNOSIS

It is impossible to predict which cases of head injury will have post-traumatic convulsions While the severity of the injury plays an influential rôle, it does not follow that the patient with the most severe head injury is most prone to the development of seizures. Similarly, study of large series of cases seems to indicate that the presence of convulsive seizures immediately following a head injury does not increase the incidence of delayed post-traumatic convulsions. In cases where focal scars are found and extirpated, the prognosis must always be guarded Following surgical excision, scars may again form

Chapter XV

SURGICAL TECHNIQUE

The surgical treatment of head injuries requires equipment which is not always available in the general hospital Adequate suction apparatus and a suitable electrosurgical unit are essential The usual operating table is suitable However if the surgeon has his choice a table with a head piece especially designed for cranial surgery is desirable It is prefer able to have as complete motility of the table as possible

The instruments must include a trephine or a set consist ing of a Hudson drill with perforator and burrs Various bone forceps (rongeurs) consisting of small medium, and large duckbills DeVilbiss or Montenovesi double-acting rongeurs and Cushing decompression rongeurs are valuable aids Gigli guides and saws are necessary for turning flaps Periosteal elevators and flap elevators facilitate various procedures Dural elevators and guides are required for incising the dura mater It is important also to have a silver clip set to control hemorrhage from vessels which are too large to control with electrocoagulation Finally the set should include ventricle needles and various brain retractors For subtemporal decom pression the Cushing retractors are of great value Surgical supplies include cottonoid dental rolls bone wax cellophane celluloid plates and silk sutures also saline solution and syringes for irrigation (Figs 73 74 75 76)

Needless to say adequate illumination is important. Usually even the most modern overhead light is inadequate This may be supplemented either by accessory focusing lamps or by a headlight

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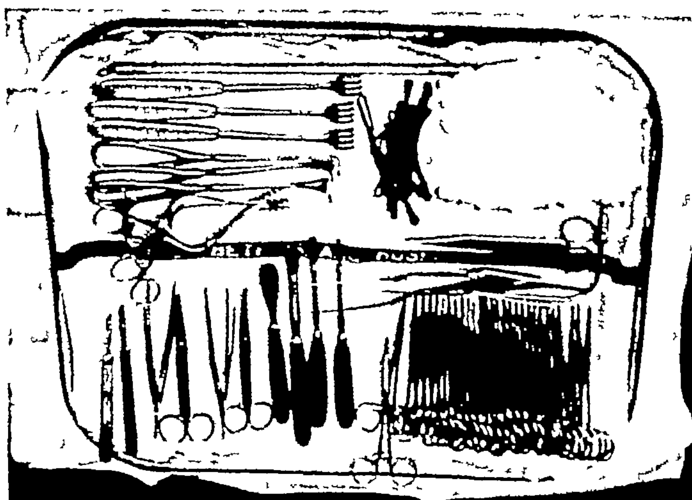


Fig 73 Instruments frequently used in cranial surgery



Fig 74 Bone instruments used in cranial operations

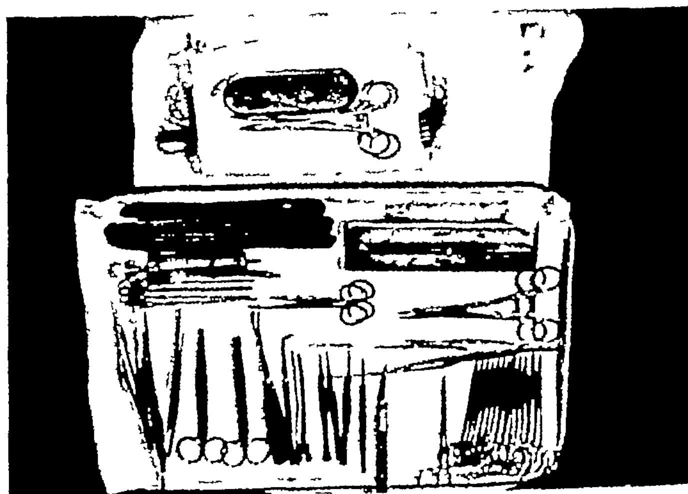


Fig 75 Dural tray

The surgical team should consist of the operator a skilled first assistant and a second assistant We stress the importance of an experienced first assistant because of the undue



Fig. 76. Arrangement of instruments and supplies on nurse s table in preparation for a cranial operation

trauma that may result from inexperienced brain retraction or unwise and unskilled suction with the brain exposed The first assistant usually performs the suction duties which require skill in the use of the suction apparatus. The surgical team is complemented by a surgical nurse trained in the handling of the materials used in brain surgery

All cranial operations must be preceded by careful preparation of the scalp The entire head is shaved closely preferably shortly before the operation so that accidental cuts do not have an opportunity to become infected After shaving, the head is thoroughly scrubbed with soap water alcohol and ether Before draping, the field is prepared with iodine and alcohol

The brain will tolerate less trauma than any other organ Brain cells that are destroyed are irrevocably lost and, therefore a thorough knowledge of cerebral topography and the physiology of the cerebral vascular system is essential if the surgeon is to avoid cellular disturbances or interference of function due to an impaired blood supply

In addition to the knowledge the surgeon already has at his finger tips as a result of experience in general surgery the following neurosurgical principles should be observed Hemostasis is of utmost importance The brain does not tolerate the loss of blood as well as other structures. By trial and error neurosurgeons have found that the most effective manner of controlling the extremely vascular scalp, in the first step of any head operation, is to have both assistants press down on the scalp on either side of the projected skin incision with their finger tips (Fig 77) When the incision is made the assistants "push down and pull apart" With good assistance the scalp wound is thus made to gape A bloodless field is obtained, and the cut edges of the galea are exposed Clamps are then placed on the galea in parallel lines, one side being clamped at a time, the assistant pressing on the scalp until the clamps can be reflected perpendicularly over the cut edge By this time the vessels of the scalp, all of which course through the subcutaneous tissues, are controlled without the necessity of clamping and tying each individual vessel. Bleeding from the bone is controlled with bone wax

Bleeding from dural vessels is best controlled either by coagulation with the electrocautery or by the use of silver clips, depending upon the type of procedure Coagulation of bleeding dural vessels tends to shrink the dura mater Where a good dural closure is desirable extensive electrocoagulation is contraindicated

Small bleeding vessels on the cortex can best be controlled with least damage to the brain by covering the area with a warm saline cottonoid wall-off More persistently bleeding vessels may require gentle coagulation, which is performed by grasping the vessel with fine pointed forceps, and then touching the forceps momentarily with the electrode Large cortical vessels are best controlled with silver clips Less dam-

age to cortical tissue results if the leptomeninges are first nicked with a sharp scalpel just beside the vessel at the point where the clip is to be placed This permits the lower half of the clip to be slipped under the vessel without traction on the surrounding meninges and cortex.

Persistent oozing from a large lacerated area in the brain can be controlled with cotton balls soaked in hot saline solution and the application of slight pressure Finally the use of muscle stamps obtained from the temporal or occipital muscle may be resorted to This is particularly useful in the control of bleeding from a tear in a sinus wall or from pacchionian granulations

The following procedures are those most commonly used in traumatic cranial surgery

1 Trephine exploration
2. Elevation of a depressed fracture
3 Subtemporal decompression
4 Osteoplastic flap

TREPHINE EXPLORATION

Trephine exploration is used in exploring for an epidural or subdural clot When bleeding from a middle meningeal vessel is suspected the trephine opening should be made in the low temporal region about 2 cm above the zygoma The most common site for subdural hematomas is the junction of the frontotemporoparietal areas on the side suspected A vertical skin incision approximately 3 cm in length is made in such a manner that if a subtemporal decompression is necessary it can be effected by lengthening the original exploratory incision The incision is carried down to the periosteum Mastoid retractors are used to hold the skin edges apart and this is usually sufficient to control bleeding from the scalp The periosteum is then incised in a cruciate

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manner and separated from the skull by means of periosteal elevators The opening in the skull may then be made either with a crown trephine, or by means of a perforator and burr When the former is used, the center pin is allowed to project beyond the level of the trephine edge, and by means of successive pronation and supination movements of the forearm and hand, a groove is made in the skull by the trephine edge The center pin is then withdrawn and the maneuver continued until the operator feels the trephine go through the inner table Irrigation should be used sparingly to keep the bone moist during trephination The button of bone is then removed Care must be exercised to avoid lacerating the dura mater The sensation on going through the inner table is unmistakable after experience is acquired. If the initial opening is made by means of perforator and burr, the former is placed in a Hudson brace and a hole then made down to the inner table The perforator is then replaced in the brace by the burr, which is used to enlarge the opening In this latter maneuver, definite resistance is encountered as the burr goes through the inner table, so that with care there is very little chance of the burr slipping through the opening and injuring the intracranial contents After the burr hole is made, the thin layer of bone usually has to be removed with a joker or with forceps

When the initial opening is made in the skull, an epidural hemorrhage can be recognized by the typical dark clotted blood which tends to herniate through the opening. In such cases the exploratory trephination is enlarged with rongeurs to about the size of a silver dollar.

When extradural blood is not found the dura mater is exposed as soon as the opening in the skull is completed In the presence of a subdural hematoma the dura mater has a plum-blue color and does not pulsate The dura mater is

then opened care being exercised to avoid injuring the underlying leptomeninges and cortex. This is best accomplished by making a superficial incision in the outer layer with a sharp pointed scalpel (such as a Bard Parker No 11 blade) The outer layer of the dura mater is then grasped with mosquito forceps and by traction the dura is raised away from the underlying leptomeninges and cortex Both layers of the dura mater may then be incised The opening is then enlarged to expose an area of cortex the size of the bony opening

The greenish-blue outer membrane of the subdural hematoma is disclosed on opening the dura mater This membrane is incised and the contents of the hematoma are removed by suction and irrigation Following this the brain usually expands to obliterate the subdural space If the subdural hematoma consists of firm clot the exploratory opening must be enlarged to permit its removal with brain spoons curettes suction and irrigation A rubber tube drain is placed under the dura mater and the wound closed in layers with interrupted silk sutures

ELEVATION OF DEPRESSED SKULL FRACTURE

The procedure employed in the elevation of simple depressed skull fractures depends upon the type of depression Every effort should be made to save the bone fragments unless they are contaminated or badly comminuted In the usual type of depression a relatively small scalp incision suffices This should be made over the border of the depression A small bony opening is then made to one side of the depression to permit the introduction of an elevator under it If there is an exposed ridge of bone at the border of the depression the opening can be made by repeated small bites with rongeurs A flap elevator is then engaged through the

opening and the depressed fragment carefully lifted back into place Closure is completed with closely placed interrupted silk sutures for the galea and for the skin Drainage must not be used

SUBTEMPORAL DECOMPRESSION

A 7 cm linear incision is made extending upward and backward from a point 1½ cm. anterior to, and a similar distance above the tragus of the ear (Fig 78) Hemostasis is obtained by tourniqueting the area with the assistants' fingers (Fig 77). Clamps are then placed on the galea and reflected, thus freeing the assistants' hands (Fig 79) A similar incision is then made in the temporal fascia It is wise to make this ½ to 1 cm posterior to the skin incision to permit imbrication during closure The fascia is then freed from the underlying muscle in order to gain more mobilization and facilitate its closure A similar incision is then made through the temporal muscle down to the squamous portion of the temporal bone This incision is made directly opposite the skin incision, and if the obliquity of the original incision is correct, the muscle incision is in line with the muscle bundles, and usually attended by very little bleeding The muscle is separated from the bone with periosteal dissectors until a bony area 7 cm in diameter is exposed. A hole is then made in the temporal bone with a trephine or a perforator and burr This opening is best made in the upper posterior part of the wound to prevent injuring the middle meningeal artery or one of its branches, since the control of its bleeding is difficult at this stage.

The skin, subcutaneous tissue and muscle are retracted with Cushing decompression retractors or self-retaining retractors, and the opening in the bone is enlarged with ron-

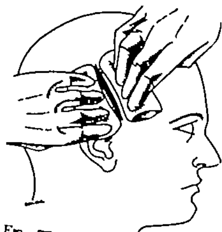


Fig. 77 Method of controlling hemorrhage from the scalp in cranial operations.

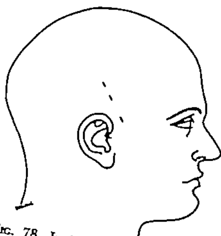
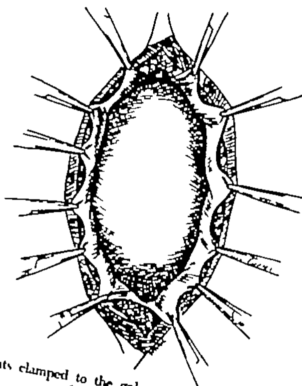


Fig. 78 Incision for subtemporal decompression.



9 Hemostats clamped to the galea control bleeding The temporal fascia is exposed.

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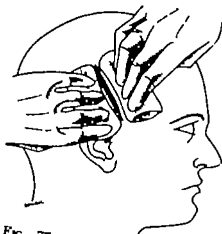


Fig 77 Method of controlling hemorrhage from the scalp in cranial operations.

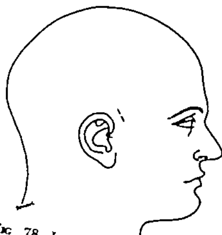


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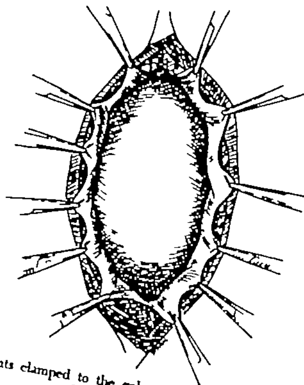


Fig. 79 Hemostats clamped to the galea control bleeding. The temporal fascia is exposed.

geurs (Fig 80) In biting downward toward the base of the middle fossa, the middle meningeal artery and its branches should be watched for Frequently the vessel is encountered

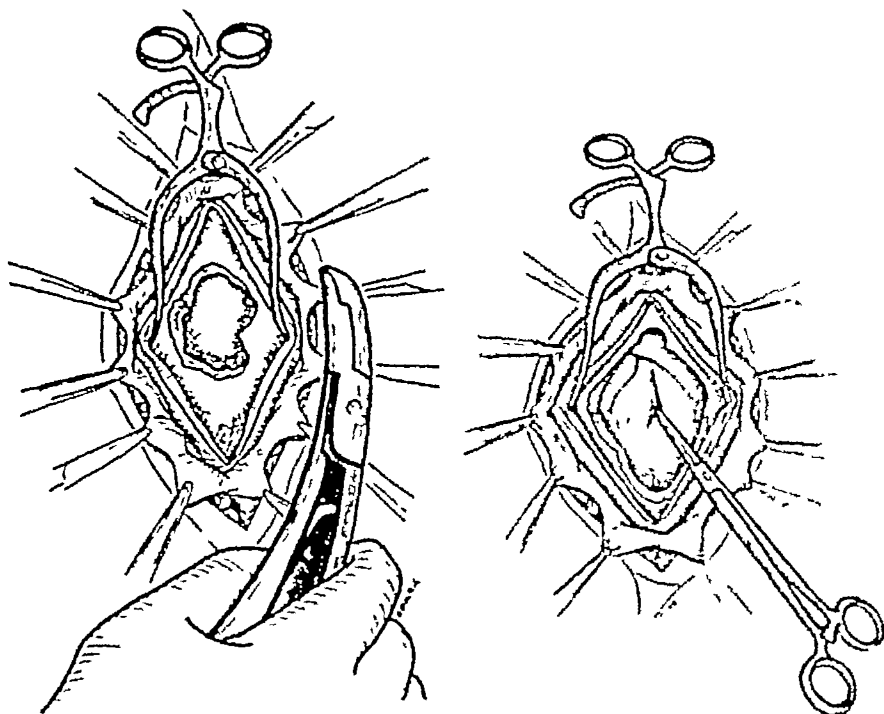


Fig 80 Temporal muscle and fascia retracted with self-retaining retractors The opening in the skull made with the trephine or Hudson drill is enlarged with rongeurs

Fig 81 The bony opening is completed exposing the dura This is opened by carefully incising the outer dural layer with a sharp pointed scalpel and then grasping and retracting the cut edge away from the cortex with a small mosquito clamp The dural opening is then completed by incising the inner layer after which a blunt dural guide can be passed and the incision enlarged in all directions

in a tunnel in the bone, and bleeding from it can be controlled with bone wax After a bony opening approximately 7 cm in diameter is made, the edges are evened as well as possible with small bone forceps (Fig 81). The dura mater is then examined The absence of pulsation denotes increased intracranial pressure The dura mater is palpated before it is

opened A drum like tension should put the operator on guard Under such circumstances opening the dura mater may result in herniation and rupture of the cortex. The ten

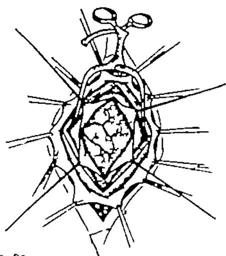


FIG. 82. The dura is opened and retracted with stay sutures.

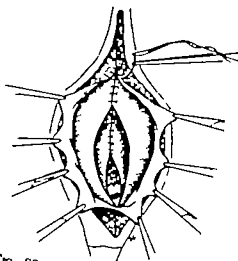


FIG 83. The wound is closed in layers with fine black silk using a layer for the muscle, one for the fascia one for the galea, and one for the skin.

sion may be relieved by one or a combination of the following procedures

1 Ventricular tap
2. Intravenous hypertonic solutions
3 Intramuscular or intravenous caffeine sodiobenzoate
4 Lumbar tap

A ventricular tap can be performed by making a small cross incision in the upper posterior aspect of the exposed dura mater The underlying gyri are usually faintly discernible through the tense dura mater and the dural incision should be made over the center of a gyrus in order to insert the brain cannula through a relatively avascular field. A knowledge of the ventricular system is essential if undue

brain damage is to be avoided. If the brain cannula is inserted upward, backward, and inward the ventricle may be reached at the junction of the temporal and posterior horns with the body of the ventricle The fluid is allowed to escape slowly. Its pressure may be measured by attaching a water manometer.

When the dura mater is not under tension it is opened widely in a stellate manner (Fig. 82), using care to avoid injury to the brain and its vessels. Since the dura mater is to be left wide open, bleeding from the dural vessels can be controlled by the electrocautery. Sponging of the dura mater and brain is done with cotton pledgets moistened in warm saline

When all bleeding is controlled, the wound is closed in layers (Fig 83), using interrupted black silk for muscle, fascia, galea and skin The most important layer in preventing unsightly herniation is the fascial layer, and special care is taken to insure a firm closure of this layer.

OSTEOPLASTIC FLAP

The projected flap is outlined on the prepared scalp by scratching with the point of a scalpel. The flap is outlined so that the base contains an adequate arterial supply. In most cases the base is in the temporal region, assuring an adequate blood supply. The incision is made in stages, the assistants controlling hemorrhage by pressure of the finger tips on either side of the incision. Usually three stages are required to complete the scalp incision Clamps are used on the distal galea. It will be found of mechanical advantage to apply Michel clips to the flap side of the incision. This will make it easier to handle the reflected flap, since when clamps are used on the flap side they must be supported and kept out

of the way by an assistant With the scalp incision made the periosteum is incised and freed from the bone with periosteal elevators For the average flap five burr holes or trephine openings are the ideal number These are so placed that a guide can easily be passed to connect them Two of the holes in the bone are made at the extreme ends of each limb of the flap The base of the flap is then prepared for fracture by biting mesially for a distance of 1 to 2 cm from each of these holes with Montenovesi or DeVilbiss bone forceps If bleeding occurs it may be controlled by packing with gauze or bone wax or both The bone flap is then completed by connecting the burr holes with saw cuts This is accomplished by passing a saw guide between consecutive burr holes, attaching the Gigli saw to the guide and drawing it through Handles are then attached to the ends of the saw and the bone cut through During the sawing the guide should be left in place between the bone and the dura mater to avoid injury of the latter structure The saw cut is made at an acute angle to the plane of the skull so that a beveled edge results This permits the flap to be replaced securely When the saw cuts are completed the flap is ready for elevation The coordination of the team is essential at this point to prevent undue loss of blood. The second assistant places his knuckles firmly across the base of the flap The operator then places a flap elevator under the flap along the limb nearest the first assistant who supports it while the operator places a second elevator under the flap at its vertex and pries the flap open It may be necessary to grasp the flap with the fingers in order to fracture the bone flap at its base In doing this the operator should be on the watch for adhesions between the dura mater and the bone if they are present they must be separated to prevent laceration of the dura mater As the flap is broken the middle meningeal artery or one of

brain damage is to be avoided If the brain cannula is inserted upward, backward, and inward the ventricle may be reached at the junction of the temporal and posterior horns with the body of the ventricle The fluid is allowed to escape slowly Its pressure may be measured by attaching a water manometer

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When all bleeding is controlled, the wound is closed in layers (Fig 83), using interrupted black silk for muscle, fascia, galea and skin The most important layer in preventing unsightly herniation is the fascial layer, and special care is taken to insure a firm closure of this layer

OSTEOPLASTIC FLAP

The projected flap is outlined on the prepared scalp by scratching with the point of a scalpel The flap is outlined so that the base contains an adequate arterial supply In most cases the base is in the temporal region, assuring an adequate blood supply The incision is made in stages, the assistants controlling hemorrhage by pressure of the finger tips on either side of the incision Usually three stages are required to complete the scalp incision Clamps are used on the distal galea It will be found of mechanical advantage to apply Michel clips to the flap side of the incision This will make it easier to handle the reflected flap, since when clamps are used on the flap side they must be supported and kept out

of the way by an assistant With the scalp incision made the periosteum is incised and freed from the bone with periosteal elevators. For the average flap five burr holes or trephine openings are the ideal number These are so placed that a guide can easily be passed to connect them Two of the holes in the bone are made at the extreme ends of each limb of the flap The base of the flap is then prepared for fracture by biting mesially for a distance of 1 to 2 cm from each of these holes with Montenovesi or DeVilbiss bone forceps If bleeding occurs it may be controlled by packing with gauze, or bone wax, or both The bone flap is then completed by connecting the burr holes with saw cuts This is accomplished by passing a saw guide between consecutive burr *holes attaching the Gigli saw to the guide and drawing it* through Handles are then attached to the ends of the saw *and the bone cut through During the sawing the guide* should be left in place between the bone and the dura mater, to avoid injury of the latter structure The saw cut is made at an acute angle to the plane of the skull so that a beveled edge results This permits the flap to be replaced securely When the saw cuts are completed the flap is ready for elevation The coordination of the team is essential at this point to prevent undue loss of blood The second assistant places his knuckles firmly across the base of the flap The operator then places a flap elevator under the flap along the limb nearest the first assistant who supports it while the operator places a second elevator under the flap at its vertex and pries the flap open It may be necessary to grasp the flap with the fingers in order to fracture the bone flap at its base In doing this the operator should be on the watch for adhesions between the dura mater and the bone If they are present they must be separated to prevent laceration of the dura mater As the flap is broken the *middle meningeal artery or one of*

its branches may be torn, and it is urgent that any bleeding be controlled at once This is usually best accomplished with silver clips Pacchionian bleeding is temporarily controlled by pressure, after covering the oozing points with cottonoid wall-offs Bleeding from bone is controlled with bone wax The fractured rim of the flap is then evened with rongeurs The entire bone and scalp flap is then wrapped in saline-soaked gauze and reflected away from the exposure Acute angulation of the scalp should be guarded against in order to insure adequate blood supply to the flap during the operation

Attention is next directed to the dura mater The pacchionian oozing is best controlled by electrocoagulation, or if the bleeding is more profuse, placing of muscle stamps is advisable Bleeding from small vessels is usually easily stopped with mild momentary electrocoagulation The dry dura mater should be carefully inspected and palpated for abnormalities Changes in color or tension are significant of underlying abnormalities If the dura mater is extremely tense, incising it may result in herniation and rupture of the underlying cortex, and for this reason effort should be made to diminish the intracranial tension by intravenous hypertonic fluids, caffeine sodium benzoate, or ventricular puncture It may be necessary to resort to lumbar puncture When the dura mater is not tense it should be opened widely, care being exercised to avoid injuring the cortex or cerebral vessels This can be accomplished by the use of dural guides and protecting, saline-soaked, wall-offs In reflecting the dural flap one must search carefully for corticodural adhesions since sudden traction on them may cause cortical injury and bleeding difficult to control

The brain is then carefully inspected The color and size of the gyri are noted With intracerebral clots the overlying

gyri are widened and darkly discolored The suspicious area is gently palpated and differences in consistency and resist ance noted. The character of localized pathological proc esses can further be determined by needle exploration The overlying pia and cortex is nicked and a cannula inserted Change in resistance is noted A cystic lesion is easily de-tected by the escape of fluid from the cannula

Having ascertained the depth and something of the char acter of the lesion, it is ready for further surgical attack by transcortical incision This incision should be made where the lesion is nearest the surface of the cortex. By making it through the center of a gyrus, bleeding is minimized First the pia mater and cortex are gently coagulated along the line of the projected incision The incision is then made, if possible at right angles to the central sulcus to avoid pres sure on the motor area in brain retraction The electric scalpel is used to make the incision which is extended down to the lesion by blunt dissection with fine pointed forceps or brain retractors

Intracranial clots can be evacuated by gentle traction with blunt forceps or pituitary forceps The clot may be lamel lated. It is usually surrounded by a brownish glial membrane which may appear at operation like tumor tissue, and make the operator feel that he is dealing with a hemorrhage into a tumor However the brain tissue beyond the membrane appears unlike tumor tissue, and subsequent microscopic examination shows it to be a glial reaction to the hemorrhage, containing considerable blood pigment.

When the clot has been completely removed and a biopsy specimen of the capsule taken for microscopic examination (to rule out tumor) all bleeding points are carefully con trolled and the wound closed. The dura mater is closed with interrupted fine black silk sutures When this closure is com

plete, the dura mater will usually be found to be relaxed and it is wise to anchor it against the inner layer of the skull by placing several sutures between it and the galea at the burr holes. This will lessen the incidence of postoperative extradural hemorrhage. The bone flap is then replaced and closure completed using interrupted fine black silk sutures for galea and skin. We prefer to leave a split rubber tube drain between the dura mater and bone, emerging from the wound through one of the posterior burr holes. The drain is removed after twenty-four hours. Careful closure of the galea is essential. The sutures should be placed about 1 cm apart. The skin sutures should be tied fairly loosely to permit some slipping, in order to prevent tissue strangulation when the scalp becomes edematous. The skin sutures are usually removed after forty-eight hours.

In craniotomy for post-traumatic adhesions or porencephaly, extreme caution must be exercised in reflecting the bone and dural flaps and in separating dural and leptomeningeal adhesions, so that undue trauma and hemorrhage may be avoided. All adhesions are carefully separated. Post-traumatic seizures in these cases are lessened or cured by carefully freeing the adhesions between the cortex and the dura mater. Electrocoagulation should be used sparingly. If a porencephaly is present, some neurosurgeons feel a communication should be established between the ventricular system and subarachnoid space through the porencephalic cyst. Often a strip of cellophane is placed over the cortex which has been adherent to the dura mater, in an effort to prevent the adhesions from recurring. Frequently the dura mater may have a large defect, and in these cases the defect should be repaired with fascia obtained from the temporal muscle or from the thigh. Occasionally, one may have to perform a craniotomy for post-traumatic Jacksonian attacks. The in-

volved area is exposed and the existing cause removed The latter may be adhesions or degenerated brain When adhesions are present they should be removed If the area is yellowish a subpial resection should be carried out If no gross pathological change can be seen the excitatory area can be localized by electrical stimulation (the area is found which on electrical stimulation provokes the characteristic attack which the patient experienced clinically), and excised.

In osteoplastic craniotomies performed to repair dural tears in fractures involving the paranasal sinuses with rhinorrhea and pneumencephaly, the scalp incision may be altered somewhat as suggested by Frazier In this type of osteoplastic craniotomy the scalp incision is a triangular one The triangular scalp flap is dissected off the bone and reflected anterolaterally The bone flap can then be made in the usual manner and reflected laterally In transfrontal flaps x ray examination must be made to measure the frontal sinuses and these sinuses must be avoided in forming the bone flap The dura mater is then examined for tears If the tear or tears are of such a character and in such a position that suture is possible this should be done with fine silk. If simple suture is not adequate, the defect should be repaired with fascia obtained from the temporal muscle

In closing the transfrontal flap an effort should be made to suture the periosteum as carefully as possible Hemorrhage should be controlled on the under surface of the scalp before it is sutured or healing may be delayed Unless meticulous hemostasis is obtained it is best to leave a drain between the scalp and bone for twenty four hours

VENTRICULOGRAPHY

The replacement of ventricular fluid by air is done with the brain cannula in a dependent portion of the ventricular

system The technique for frontal and occipital ventriculography will be given

In all cases it is best to prepare the entire head



FIG 84 Patient in position for frontal ventricular puncture The cerebellar head-rest is in the reverse position (Gross and Ehrlich, *Archives of Surgery*, 39 122, 1939)

Frontal Ventriculography

The patient is placed in the prone position with the head extended in the cerebellar head-rest which is reversed for this procedure (Fig 84) A 3 cm skin incision is made just within the hair line (10 to 12 cm above the glabella), about 2 cm. from the midline and parallel to it.

The skin and subcutaneous tissue is retracted with mastoid self-retaining retractors A burr hole or trephine opening is made in the bone The dura mater is then opened with a cruciate incision, avoiding injury to the cortex or blood vessels The pia mater is nicked in the middle of a gyrus and

a brain cannula inserted on a plane parrallel to the falx and at right angles to the skull The cannula usually easily enters the ventricle at the junction of its body with the frontal horn

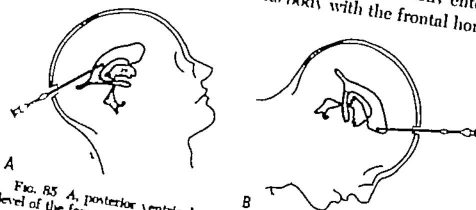


FIG. 85 *A*, posterior ventricular puncture The ventricle is tapped at the level of the foramen of Monro. *B* frontal ventricular puncture The ventricle is tapped below the foramen of Monro (Gross and Ehrlich, *Archives of Surgery* 39 122, 1939)

The size of the ventricle can usually be roughly estimated by the depth at which it is entered and the amount of fluid which escapes A normal ventricle is reached at a depth of 4½ to 5½ cm

When the ventricle is punctured in the manner described the mouth of the cannula is lower than the foramen of Monro (Fig 85) Unless there is an obstruction only one ventricle need be punctured. Good visualization of the entire ventricular system including the lateral ventricles the third ventricle aqueduct of Sylvius and fourth ventricle is obtained by this method. The exchange of air for fluid should be made fairly slowly in divided amounts We prefer an interchange in 10 c c quantities When air escapes after its injection the procedure is terminated

With the interchange completed the cannula is slowly withdrawn Slow withdrawal permits the cerebral tissue to close the cannula tract and prevents the escape of air through it.

Closure is done with fine black silk for galea and skin. The dura mater is left open The patient is then taken directly to the x-ray department where the films are taken

Posterior Ventriculography

In posterior ventriculography, the patient is immobilized on the operating table in a supine position with the head sharply flexed by means of a dental type of head-piece placed against both mastoid bones The burr holes are then placed 7 cm above the external occipital protuberance and 3 cm to either side of the midline (In right-handed individuals the first opening is made on the right side) The skin incision should be a verticle one in order to obtain least hemorrhage. After the dura mater is opened and the pia mater nicked with a sharp-pointed scalpel in an avascular area, the cannula is inserted with its point directed toward the glabella The ventricle is usually entered at a depth of 6 to 8 cm , at the junction of the posterior horn and the body of the ventricle The interchange of air and fluid is then carried out To empty adequately the ventricular system, it may be necessary to lower the head of the table in order to depress and make more dependent the side where the cannula entered the ventricular system As in frontal ventriculography, if there is any question as to communication between the ventricles, bilateral ventriculography should be performed With the completion of the interchange of air for fluid, the procedure is terminated in a manner similar to that described for frontal ventriculography.

ENCEPHALOGRAPHY

The procedure consists of the spinal insufflation of air into the cerebrospinal fluid pathways Preparation of patient for encephalography includes

1 Local anesthesia in adults

2. Avertin anesthesia in children or noncooperative adults
3. Enema the previous night

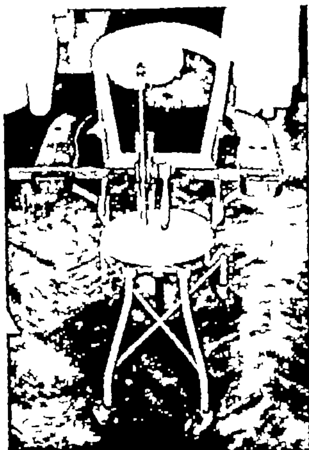


Fig. 86. Chair for encephalography

4. No meal immediately preceding the procedure Nothing by mouth for four hours prior to encephalography
5. Atropine 0 00075 Gm (gr 1/80) by hypodermic injection 15 minutes before

The procedure is carried out in the department of radiology The patient is seated on a bench with the forehead resting against a verticle Potter Bucky diaphragm If a special chair is available (Figs 86 and 87) the procedure is simplified and the patient is more comfortable A lumbar

puncture is performed with the patient in this position Only one needle is inserted and a reading of the cerebrospinal fluid pressure is obtained by attaching a water manometer

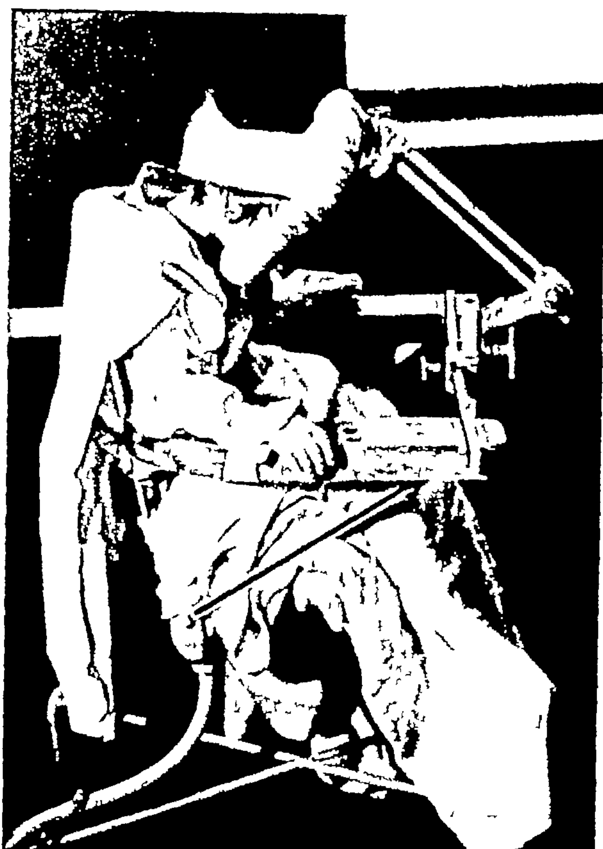


FIG 87 Encephalographic chair in use The patient's head fits comfortably in the cerebellar head-rest which was adapted to the chair by means of the metal cross bar

It is unnecessary to repeat pressure readings because it has been shown repeatedly that at no time during the procedure is the original pressure exceeded except in response to coughing or straining Air or oxygen may be used for injection. When encephalography is only occasionally performed, it is just as well to use air since the use of oxygen requires slightly more elaborate equipment, and the difference be-

tween the two is not marked enough to warrant the additional expense

Atmospheric air is sucked into a sterile 20 c c syringe through many thicknesses of sterile gauze in order to avoid possible contamination by air borne particles The first 10 c c of cerebrospinal fluid is slowly removed. The fluid is never sucked out but is allowed to push the plunger out of the syringe by its own pressure The syringe is never attached directly to the needle but the two are connected by a piece of rubber tubing 4 cm in length This makes the connection flexible, and prevents displacement of the point of the needle during the procedure. By compressing the tube with the thumb and forefinger the use of a stopcock is avoided After the removal of the first 10 c.c. of fluid 5 c.c of air is injected at approximately the same rate of speed as the fluid escaped The interchange of the air and fluid is then carried out with 5 c.c of each at a time.

After 20 c c of air have been injected postero-anterior x ray films of the skull are taken and sufficient air is usually present in the ventricles to permit an estimation of their size The procedure is continued as the film is being developed and it is usually ready for viewing by the time 50 to 60 c c of air have been injected When the ventricles are of normal size 65 c.c of air is generally found to give suitable encephalograms Large ventricles require more air marked hydrocephalus is usually well visualized after the injection of 120 to 140 c.c. of air It is not necessary to empty the cerebrospinal fluid passageways since the replacement of the estimated 75 c c of fluid in the spinal canal does not aid in securing better encephalograms.

The x ray exposure should be made in the upright position immediately after the injection of air Antero-posterior postero-anterior and lateral stereoscopic views of each side

should be taken and then the entire procedure repeated with the patient in the horizontal position With this series of films good visualization of the entire ventricular system

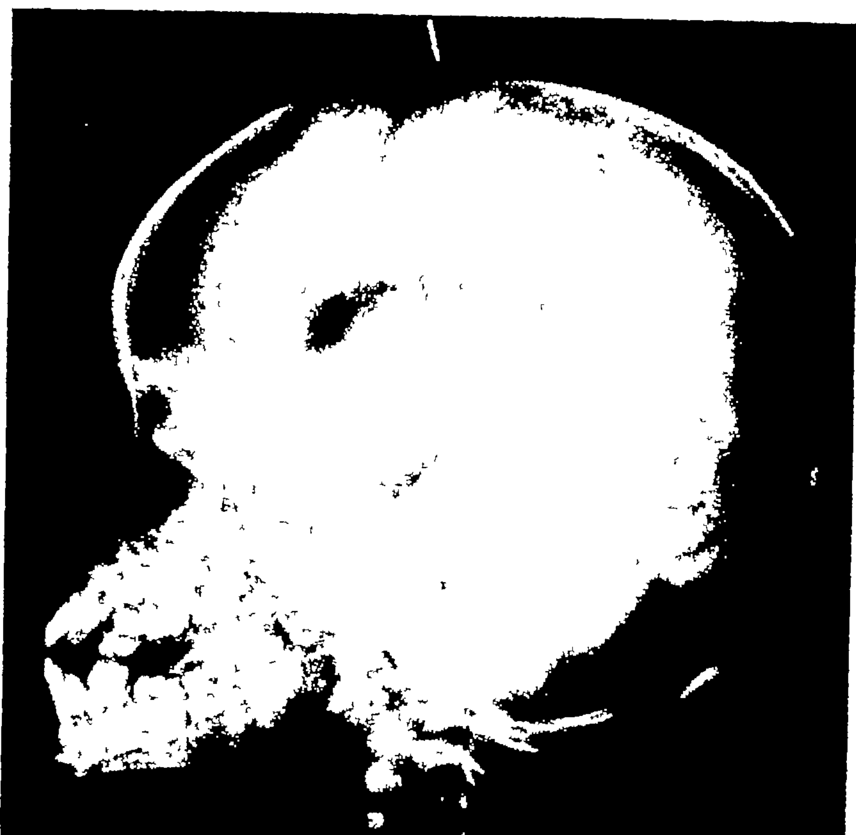


Fig 88 Normal encephalogram Note lateral ventricle, third ventricle, aqueduct of Sylvius, and cisterna magna

(Fig. 88), the cerebral sulci and subarachnoid cisterns can be obtained, and special posturing is not necessary

ANESTHESIA

The type of anesthetic drug and its method of administration are of paramount importance in carrying out surgical procedures upon patients with acute head injuries In most instances circumstances prevent complete preparation or

study of the patient the time element frequently being an important factor in the prognosis The anesthetic drug chosen must therefore be safe for all patients regardless of age or preexisting organic disease

The method of administration too must be attended with minimal risk, not only during the operation but also in regard to complications during the postoperative period Since inhalation anesthesia is accompanied by asphyxia and excitement during induction it is contraindicated

It is evident, then that local anesthesia is the method of choice whenever possible. In comatose or deeply stuporous patients it is a simple matter to block the operative field by injecting a 1 per cent procain solution or some other anesthetic drug into the surrounding subcutaneous tissues This should be done even if the patient is in profound coma at the time of operation since in favorable cases the state of consciousness may so improve during the course of the operation that painful stimuli may be perceived

In restless or uncooperative persons preoperative sedation or basal anesthesia may be required before the local anesthetic solution is injected. Sodium pentobarbital (nembutal) in doses of 0.2 to 0.3 Gms (gr 3 to gr 4½) given about one and one-half to two hours before the time of operation is an excellent drug for this purpose When more rapid action is desired, tribromethanol (avertin) may be given by rectum This drug is so rapidly absorbed that its effects are evident within a very short period It should be employed only as a basal anesthetic, the dose not exceeding 80 mg per kilogram of body weight and its preparation and administration entrusted to one who has had experience with its use

The sensory nerves supplying the scalp are derived from the trigeminal nerve and cervical plexus (Chapter I) These

nerves emerge from the galea and become subcutaneous approximately along a line extending from the glabella to the external occipital protuberance. The anesthetic solution should therefore be injected into the subcutaneous tissue surrounding the operative field, and not below the galea. In the temporal and suboccipital regions where the skull is covered by muscles, the anesthetic solution must be injected into the muscles to obtain complete anesthesia

One per cent procain solution is used routinely Ordinarily the solution is made up without the addition of adrenalin, since the latter drug may cause a marked rise in blood pressure.

For débridement of scalp lacerations or compound skull fractures, or the elevation of depressed skull fractures, the anesthetic solution is injected in a narrow band surrounding the operative field For subtemporal decompression the injection is made in the line of the proposed incision and into the temporal muscle

Chapter XVI

COMPLICATIONS AND SEQUELAE OF HEAD INJURIES

Other complications of head injuries not previously discussed may be divided into the early and the late, the frequent and the unusual The psychiatric aspect of head injuries requires a volume by itself and only a brief outline can be presented in a work devoted to the acute phases of head injuries.

Among the early but more or less unusual complications are traumatic arteriovenous aneurysm of the internal carotid artery spurious traumatic meningocele diabetes insipidus and subcutaneous emphysema.

ARTERIOVENOUS ANEURYSM OF THE INTERNAL CAROTID ARTERY

Fractures of the middle cranial fossa extending into the sphenoidal fissure may produce a tear in the internal carotid artery as it traverses the cavernous sinus A direct communication between the arterial and venous systems is thus established and results in an arteriovenous aneurysm or fistula The symptoms depend upon the lesion itself the proximity of the optic oculomotor trochlear and abducens nerves and the ophthalmic division of the trigeminal nerve and also to the interference with venous return from the ophthalmic vein which empties directly into the cavernous sinus

The lesion itself is productive of a swishing intracranial bruit synchronous with the pulse This distressing symptom

following a head injury is a clue to the diagnosis. The bruit can be heard with a stethoscope placed over the temporal region or over the eye on the affected side, and can be obliterated by pressure on the common carotid artery in the neck The eye signs are of great importance The globe pulsates and is proptosed (pulsating exophthalmos) There is usually marked injection of the conjunctival veins as well as the veins of the eyelids Ocular movements are greatly diminished or absent depending upon the degree of impairment of third, fourth, or sixth nerve function Ophthalmoscopic examination reveals marked congestion of the retinal veins and swelling of the optic disc

The treatment of traumatic arteriovenous aneurysm consists of digital compression of the carotid artery followed by ligation At first the artery is compressed for a few minutes every three or four hours. The compression is gradually prolonged When the patient is able to tolerate compression for an hour three or four times a day, the artery may be ligated This should always be done under local anesthesia so that the state of consciousness and power of the opposite upper and lower extremities can be watched If any untoward signs or symptoms develop, the ligature is cut at once If preliminary compression has been carried out long enough, permanent ligation of the common or internal carotid artery is safe A double-waxed silk suture is used for the purpose Following this the bruit ordinarily disappears at once and the other symptoms gradually clear up.

SPURIOUS TRAUMATIC MENINGOCELE

A subcutaneous collection of cerebrospinal fluid following a fracture of the skull is known as a spurious traumatic meningocele (Fig 89) It occurs without laceration of the scalp

but with a tearing of the dura mater by a bone fragment Evidence of a fracture of the vault and the presence of fluid under the scalp are diagnostic. In many cases it is difficult



FIG. 89 Spurious traumatic meningocele This gradually cleared up without aspiration or other interference

to differentiate spurious meningocele from hematoma, though in the latter there is usually a surrounding raised edge Collections of fluid under the scalp should not be incised or aspirated since they are absorbed sooner or later and intervention invites infection

TRAUMATIC DIABETES INSIPIDUS

Rarely following head injury the patient develops an excessive thirst and excretes large amounts of urine of a low specific gravity This unusual disturbance of water metabolism is due to injury of the hypothalamus which is more likely to be traumatized in fractures of the middle cranial fossa Injections of pituitrin control the polydipsia and polyuria

Repeated hypodermic injections of 1 c c. are needed as long as the disturbance exists

SUBCUTANEOUS EMPHYSEMA

Subcutaneous emphysema is unusual after head injuries. This phenomenon may occur when a fracture of the skull extends into the frontal or ethmoid sinuses and permits air to escape into the subcutaneous tissues of the forehead and face It can be differentiated from gas bacillus infection by its appearance immediately after the injury and its benign course The air is absorbed from the subcutaneous tissues spontaneously

TRAUMA AND MENTAL DISEASE

Mental aberrations frequently accompany head injuries In most cases they are of a temporary nature and clear up promptly when the state of consciousness returns to normal Complete amnesia for the accident and the events preceding it (retrograde amnesia) may occur In a severe injury, as consciousness returns delirium often replaces coma Confusion, resistiveness, and marked restlessness which is frequently refractory to all forms of sedation, accompany this acute traumatic delirium It usually clears up in a few days, but in some cases may persist for a month or longer The patient may remain confused and disoriented for many weeks During his wakeful periods he may be very noisy and resistive At times it is difficult to keep clothes on such a patient Incontinence is a frequent complication Occasionally the delirium and the restlessness are so severe that the patient dies of exhaustion. The treatment of these symptoms often taxes the resources of even the most experienced therapist Most important is adequate and skillful nursing care. External stimuli should be reduced to a minimum. The

tendency of attendants and members of the patient's family to talk to him must be curbed Everything must be done to insure the patient's comfort. The room should be darkened and properly ventilated The patient's bowels bladder and skin must be properly attended to Restraints should be avoided unless they are absolutely necessary to prevent the patient from harming himself Sometimes heroic measures are justified and the administration of avertin may be required to control a patient who would otherwise exhaust himself When these patients recover they have an amnesia for the period during which they were delirious and confused They are at first completely disoriented Frequent mood fluctuations occur so that euphoria and depression alternate in rapid succession Excitement in some cases is so active as to border on mania.

This type of patient presents the greatest difficulties in treatment. Because of the marked restlessness maintenance of nutrition and fluid balance may become a serious problem In these as in other cases of head injury the guiding principles are (1) avoidance of external stimuli by keeping the patient in a quiet and darkened room (2) the use of appropriate sedatives, such as the barbiturates paraldehyde, chloral and bromides In extreme cases it may be necessary to use avertin to quiet the patient enough to administer intravenous fluids and stomach tube feedings. In the absence of evidences of kidney or liver damage avertin may be used repeatedly without deleterious effects

POST TRAUMATIC SYNDROME

Many head injuries are followed by a train of subjective somatic symptoms accompanied by personality changes and mild mental disturbances The incidence of this troublesome sequela is influenced by several factors The severity of the

injury may play some part, since post-traumatic symptoms develop more frequently after severe injuries than after mild ones The age of the patient at the time of the injury is of great significance Persons over 50 years of age who suffer head injuries nearly always develop post-traumatic symptoms, and these symptoms persist longer than in younger persons

Symptoms may not be apparent until the patient is up and about In his contacts with his family and friends the personality changes are soon noted. Increased irritability, emotional instability, intolerance, moodiness, difficulty in concentration, and absent-mindedness are causes of suffering to the injured person and to those with whom he comes in contact Headache occurs more often than any other of the subjective somatic symptoms Some individuals describe the headache as constant while others complain of it only upon mental or physical effort

Dizziness is the next most frequent symptom Some patients have a constant sensation of dizziness or giddiness, others complain of dizziness only when changing position, or when looking upward Rapid fatigability which lessens or abolishes the injured person's capacity for work, and intolerance to alcoholic beverages are often present

Neurological examinations do not usually disclose marked abnormalities Careful and painstaking studies, however, may show minor, yet nevertheless, definite alterations Slight inequality of the pupils, disturbances in convergence, slight facial weakness, abnormalities in the vestibular tests, and slight reflex inequalities may be found Laboratory studies other than roentgenograms of the skull taken after lumbar air injection are of little help in diagnosis.

Psychological investigations carried out on many patients

recovering and convalescing from acute head injuries show that even among those with no apparent mental defect clinically there is a demonstrable impairment in mental



FIG. 90 Encephalogram from a patient who was operated upon for a head injury several years ago At the time of the encephalographic study he had a right hemiparesis and some disturbance in speech. Note the asymmetrical dilatation of the ventricles with the larger one on the side of the lesion.

processes particularly evident when the patient is required to perform tasks necessitating abstract thinking

Encephalographic studies after head trauma may provide visible evidence of structural changes in the brain even in the absence of abnormal neurological signs A small propor-



Fig 91 Lateral view of Figure 90

tion of patients are markedly improved by encephalography Symmetrical enlargement of the lateral and the third ventricle is the most common encephalographic finding Asymmetrical dilatation (Figs 90 and 91) of the lateral ventricles with a shift toward the side of the lesion may accompany marked focal signs such as hemiplegia, aphasia, or hemianopsia Increased amounts of air in the cerebral sulci and large collections of air in the frontal subarachnoid space are other evidences of cerebral atrophy following trauma. While encephalographic abnormalities are of utmost diagnos[illegible] when [illegible] ir absence does not rule out or[illegible] use of the post-traumatic [illegible] scars and diffuse cortical

degeneration may not cause any distortion of the cerebral ventricles and subarachnoid spaces which can be demonstrated in the encephalogram

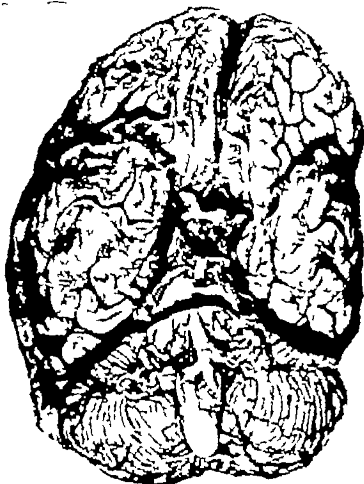


FIG. 92. Brain from a patient who had marked post-traumatic symptoms following a head injury An encephalogram done a few months before he committed suicide by hanging was within normal limits. Note areas of softening in the right orbital convolutions and the tip of the right temporal lobe

The incidence and severity of post-traumatic symptoms can probably be reduced by adequate care during the acute phase of a head injury This includes rest in bed for at least

Encephalographic studies after head trauma may provide visible evidence of structural changes in the brain even in the absence of abnormal neurological signs A small propor-



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degeneration may not cause any distortion of the cerebral ventricles and subarachnoid spaces which can be demonstrated in the encephalogram

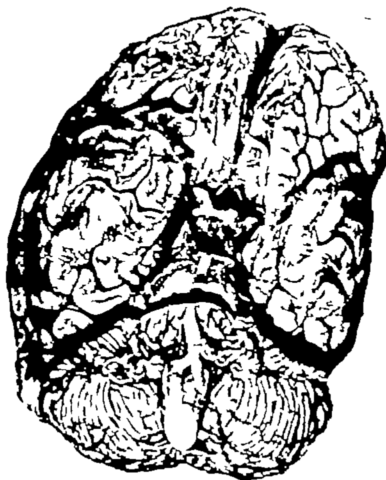


Fig 92. Brain from a patient who had marked post traumatic symptoms following a head injury. An encephalogram done a few months before he committed suicide by hanging was within normal limits. Note areas of softening in the right orbital convolutions and the tip of the right temporal lobe

The incidence and severity of post traumatic symptoms can probably be reduced by adequate care during the acute phase of a head injury. This includes rest in bed for at least

three weeks followed by a prolonged convalescent period before returning to work During this period, small doses of a mild sedative, such as phenobarbital, combined with sympathetic understanding by the physician treating the injured person, will hasten recovery In many patients the post-traumatic syndrome is colored by symptoms which seem to be of a functional nature, particularly in the presence of impending litigation and in compensation cases In addition, psychoneurotic symptoms may be precipitated when the element of fear is associated with the injury.

The combination of organic and psychological factors in the genesis of the post-traumatic syndrome adds to the difficulties in treatment. While it is true that symptoms are prolonged for financial gain in some instances, there are just as many cases where no improvement follows a satisfactory settlement, or where monetary considerations play no part Much harm can be done if the legal aspects of the injury are over-emphasized or if the patient is coerced into returning to his regular employment before the physical and psychological disturbances have become stabilized at a level permitting normal responses to the demands made on him

After a reasonable period of complete rest, the injured person must be encouraged by his own physician in cooperation with the employer to return to work At first part-time work or a lighter job may enable gradual adjustment to take place Most individuals are able to return to work within six months after a severe head injury In some cases one to three years are required for stabilization, and even then relapses may occur In about one-third of the cases permanent partial disability results, and an additional 5 to 10 per cent are totally and permanently incapacitated

The hostile attitude of many examiners for industrial in-

surance companies toward the individual with post traumatic symptoms does much harm and often prolongs secondary psychological elaborations Out-and-out malingering is uncommon and easily recognized

"PUNCH DRUNK"

In 1928 Martland described a chronic progressive disease of the brain in pugilists, which he termed "punch drunk." The syndrome is characterized by awkwardness of gait slight mental confusion slowing up of all coordinated muscular movements, and tremor of the hands and head. Later with further progress of the process, mask like facies muscular rigidities and mental deterioration develop Martland believed the disease due to multiple repeated petechial hemorrhages in the corpus striatum the result of repeated head injuries incurred by pugilists Similar injuries with similar symptoms may result from a single severe blow to the head. In the following case a clinical syndrome similar to that described by Martland in pugilists developed after a single severe trauma

Case 21 A police sergeant, 50 years old, was in an automobile collision. He was unconscious for about twenty four hours. He was in a hospital for twenty two days. When he returned home his wife noticed a marked alteration in the patient's behavior Previously alert and easy going he became slow absent minded and hyperirritable In walking he had a feeling as if he were falling over Because of these symptoms he came under our observation seven months after the accident The patient had a mask like facies Simple arithmetical problems were performed slowly and with many errors. All questions were answered after a prolonged latent period There was a right central facial palsy and the deep reflexes were all increased. Coordination tests revealed moderate ataxia in the left lower extremity The mental and neurological changes were indicative of diffuse brain damage

MENTAL CHANGES IN CHILDREN FOLLOWING HEAD INJURIES

Children are much less likely than adults to develop post-traumatic symptoms after mild or moderate head injuries. However, severe head injuries, in which the initial period of unconsciousness persists for several days or more, are frequently followed by marked behavior and personality disorders Permanent mental enfeeblement may result from severe injuries sustained during infancy or early childhood In later childhood, the personality changes may be such that a previously docile and well-mannered child becomes a difficult behavior problem often necessitating care in an institution or special school

Mental and behavior disturbances often go unnoticed until the child who has suffered a severe head injury returns to school A child, previously able to compete satisfactorily with his schoolmates, falls behind Defects in memory, attention, and deportment are prominent At home the child may complain of headaches and have explosive outbursts of temper In some cases juvenile delinquency develops after a severe head injury The following case is rather typical of the mental changes following a severe head injury in a child

Case 22 A boy 8 years old, was run over by a truck. He was admitted to the hospital in shock, bleeding from a laceration in the left occipital region All the reflexes were absent He was treated for shock External heat, caffeine, and 10 per cent glucose, injected intravenously, were administered After a few hours he began to react to the treatment The scalp wound was thoroughly débrided under local anesthesia the next day The patient remained semi-stuporous for many days He became very restless when disturbed for dressings or other therapeutic procedures His sensorium cleared gradually After four weeks he was

fully conscious but very apprehensive and restless The patient was sent home after six weeks in the hospital. An x ray film of the skull taken just before discharge disclosed an extensive linear fracture of the left parietal and occipital bones At home he became a behavior problem. He began to stutter had temper tantrums, his memory became poor and his teachers described him as an "entirely different boy" than the one they knew before his head injury The patient was followed for two and one-half years. During this time there was no perceptible improvement in his behavior

Treatment of children with behavior disturbances requires tact, patience and sympathetic understanding If the child is unable to compete with his classmates on a satisfactory level he should be placed in a special school or an ungraded class where work beyond his capabilities will not be assigned The parents must be instructed so that a satisfactory program for this child's training can be carried out. Regular hours, avoidance of excitement, and carefully supervised recreation are part of the plan Small doses of a mild sedative such as phenobarbital should be given for several months

Chapter XVII

HEAD INJURY AND DISEASE OF THE NERVOUS SYSTEM

The rôle of head trauma in the causation of disease of the nervous system is often difficult to evaluate Occasionally a direct relationship between the two is easily established, but in most diseases the part played by trauma is indefinite; experts frequently disagree when confronted with an individual case When the trauma is associated with an industrial occupation or is involved with impending litigation, the problem becomes even more difficult

Wechsler divides cases of trauma to the nervous system into several classes First, those in which the injury to the nervous system is a direct and immediate result of the accident In this group are fractures of the skull, intracranial bleeding, and contusion and laceration of the brain

In the second class are the cases in which the nervous system shows the effect of trauma a few hours, or perhaps several days, after the accident For instance, the slow middle meningeal bleeding which may give symptoms twenty-four hours or more after the trauma, the syndrome known as "late apoplexy," in which slow thrombosis of a cerebral vessel takes place and symptoms may occur a week or two after the accident Occasionally, meningitis or even brain abscess complicating a fracture may fall into this group On the whole there is usually little difficulty in establishing a relationship between the trauma and the disease of the central nervous system in such cases

In the third group are those cases in which there are no immediate effects of the accident but only late sequelae, for example, convulsions developing months or years after an injury to the brain Differences of opinion may be voiced as to the relationship between trauma and late symptoms but on the whole most observers agree that one exists

In the fourth group are those cases in which symptoms indicative of pathological changes in the central nervous system follow immediately or reasonably soon after an injury, but in which the trauma alone is not responsible This group includes the incipient undetected general paretic who receives a blow to the head and then rapidly develops all the symptoms of the disease Years ago before the spirochete was known to be the cause of paresis the pathogenesis of this disease was attributed to the trauma Now it is known that general paresis is never caused by trauma alone but that its symptoms may develop rapidly after an injury Other examples are the alcoholic individual who may show rapid deterioration following a blow to the head, a patient with marked cerebral arteriosclerosis who may develop dementia following trauma and the epileptic patient whose symptoms may be aggravated by a blow to the head, perhaps during one of his seizures It is in this group that one finds justifiable differences of opinion particularly in medicolegal cases

The fifth group includes those cases in which at indefinite intervals following an accident the victim develops psychic or mental sequelae of head injuries These cases comprise a large part of the so-called traumatic neuroses In this group the relationship of the trauma to the symptoms is a much debated question and the opinions of well-qualified experts may be at variance On the whole, it is generally accepted that there is a direct relationship. However that the influ

ence of expected compensation is a major factor, is illustrated by the many instances in which symptoms clear up after financial settlement is made. On the other hand, there are cases in which symptoms persist, and in which the financial advantage to be gained by the maintenance of symptoms is practically nil. Investigation into the pathological processes following head injury reveals that in a large number of cases, even relatively slight trauma to the head may be followed by definite organic changes, some of which are easily demonstrable on postmortem examination. These consist of numerous petechial hemorrhages and areas of softening with secondary sclerosis and dilatation of the ventricles which might account for the numerous symptoms seen in so many of the cases classified as traumatic neuroses. Furthermore, in all probability, there is a large group in which pathological changes are physiological rather than anatomical, the result of alterations in the cerebrospinal fluid mechanics or in the cerebral circulatory mechanism. In these cases no anatomical changes may be found, either at postmortem examination or by encephalography. Because of the complicated and difficult aspects of this problem, it is probably much wiser to refer to the late symptoms following head injury as the "post-traumatic syndrome."

From a medicolegal standpoint the interval between the head injury and the onset of symptoms is particularly important. Generally speaking, the longer the interval between the two, the more difficult it is to establish a relationship between them. It should be remembered that when there is pre-existing disease, such as general paresis, alcoholism, epilepsy, and so on, the disease itself may be the cause of the accident producing the injury.

From the point of view of compensability, it is obviously extremely important to determine whether the trauma caused,

precipitated, or aggravated the disease It is also necessary to determine, as well as possible the prognosis This includes the probable duration of symptoms and the expected degree of restoration of function

PARALYSIS AGITANS

Paralysis agitans (Parkinsonism) is a chronic progressive disease of the corpus striatum and globus pallidus It occurs most frequently after the fifth decade and is more common in males than females The lesions found in the brain at autopsy are usually attributed to arteriosclerosis or encephalitis Although traumatic injuries may produce similar lesions true paralysis agitans is rarely the result of head injury lesions due to trauma are usually widespread and it is only when petechial hemorrhages are concentrated in the basal ganglia that a clinical condition closely resembling true Parkinsonism ensues When the symptoms of paralysis agitans develop after a head injury and there are evidences however slight of lesions in other parts of the brain the head injury must be considered as the precipitating if not the primary etiological factor

VASCULAR DISEASE

Various types of cerebral softening and vascular lesions following trauma have been described Oppenheim for example believed that premature disease of the vessels of the brain resulted from head injury Friedman described pathological changes of the cerebral vessels and thrombosis of capillaries was demonstrated by Trousseau Such changes in the vessels can weaken the vascular walls so that the patient is predisposed to cerebral hemorrhage This is the explanation given for "late apoplexy" following trauma

Immediate intracerebral hemorrhage may of course fol

low trauma Pre-existing areas of softening will diminis
the support of the cerebral vessels, and the possibilities o
trauma causing a hemorrhage are greatly increased. Globu
and Strauss stressed the importance of this factor in favor
ing massive cerebral hemorrhage

No evidence exists that trauma is the cause of cerebra
arteriosclerosis.

NEUROSYPHILIS

Paresis The relationship between head injury and paresi
has been given a greal deal of consideration Before the tru
etiology of paresis was known, head trauma was considere
to be the chief agent Sir F M Mott, one of the pioneers i
the study of the pathology of the central nervous system
believed that general paresis was more likely to develo
after a head injury than without it. Court decisions gave
evidence that both the laity and judiciary had been con-
vinced of the relationship between trauma and paresis

In 1913 Noguchi demonstrated spirochetes in the brains
of paretic patients, thus relegating trauma to a secondary
rôle as an etiological factor Invasion of the brain by the
spirocheta pallida produces atrophy of the brain cells with
an overgrowth of glial elements Such underlying patholog-
ical changes make it impossible to assign a direct causal
relationship when the symptoms of paresis develop imme-
diately after a head injury Nevertheless, nearly all observers
agree that head trauma is frequently an important precipi-
tating factor There is investigative evidence to prove that
when spirochetes are present in the blood stream, a site of
trauma tends to be invaded by the organisms For example,
when a wound was made in a rabbit's skin, the intravenous
injection of spirochetes was followed invariably by the ap-
pearance of a chancre at the site of the wound

It is generally believed that to establish a relationship the interval between injury and the onset of symptoms of the disease should be not less than two months nor more than three years On the whole however it has been found from experience that no rigid rules can be laid down and each case must be considered on its own merits

Clinically we find many patients with serologically positive spinal fluids who remain symptom free Following a head injury such patients may develop all the signs and symptoms of general paresis It devolves upon the medical man qualified as an expert to give his opinion regarding the probabilities of the disease continuing to be latent had the injury not occurred. It is well known how widely these opinions differ in individual cases

On the whole it may be said that any trauma which is severe enough to cause organic changes in the central nervous system may be a definite contributory factor in establishing symptoms in a known luetic individual who previous to the trauma was symptom free It is generally accepted by the courts that trauma might accelerate the course of the disease and produce symptoms which otherwise might not have appeared In the absence of scientific proof to the contrary the injured individual is given the benefit of the doubt Differences of opinion are usually due to questions as to the severity of the injury necessary to accelerate the disease the time interval between the injury and the onset of symptoms or the progression of symptoms following an injury It is for this reason that the courts so frequently find that two well known and well qualified experts voice opinions which are diametrically opposed

Patients with luetic cerebrovascular disease are more prone to vascular accidents (massive cerebral hemorrhages) as a result of trauma When an inflammatory process has weak

ened the walls of a cerebral vessel head trauma may cause a rupture of the blood vessel with development of the usual symptoms of intracerebral hemorrhage That such an outcome may have occurred in due course without the trauma may be plausible from a medicolegal standpoint The courts have rendered decisions indicating that trauma is not disregarded as the cause of the hemorrhage

Gumma of the skull may follow head trauma in a luetic patient, and despite the fact that the patient was known to have syphilis before the injury, such a complication has been held to be compensable when the injury was sustained by a patient while at work.

Similarly, *luetic meningitis* developing within a reasonable time after a head injury has been considered to have been precipitated by the trauma

"Granting that the benefit of the doubt should accrue to the victim, when there is reasonable doubt as to the effectiveness of any given trauma in the production of the disease, it seems that the medical expert has the distinct duty of keeping within the limits of what is reasonable, and must not allow his imagination to run riot and give the dignity of probability to conclusions that may only be considered as remotely possible" (Solomon)

BRAIN TUMORS

The rôle of trauma in the causation of tumors of the brain has occasioned considerable controversy This problem is of great medicolegal importance, and warrants inclusion in this chapter of some of the opinions voiced by others.

Gowers, in 1888, was emphatic in his belief that trauma could cause a neoplasm. "There is little doubt that traumatic influences, falls and blows on the head, are occasionally the immediate excitants of a growth, since the symptoms have

been observed to follow a blow and the tumor is found to correspond in position to the seat of the injury Such a relationship has been observed in almost all forms of tumor, in syphilitic and in tubercular growths as well as those which are supposed to be of purely local origin Traces of traumatic mischief may or may not be visible after death and immediately the nutritive changes consequent on a mere concussion may be the starting point of a tumor But the cases in which a traumatic cause can be traced form a very small proportion of the total number, and the extent of this influence may be readily over rated"

James Ewing in his textbook says "The frequency of the traumatic origin of brain tumors has been variously estimated by different observers, but as a rule the estimates are notably high" Gerhart accepted the traumatic origin of 10 among 60 reported cases of glioma and in 4 of 11 cases of his own Bruns believes that a direct traumatic relationship is rare

Oppenheim states that trauma may indirectly excite tumor growth by establishing a condition of epilepsy which eventually induces the neoplastic proliferation of glia tissue. Such cases are also reported by P Knapp, and Ewing adds, "trauma may be the direct exciting cause of the tumor In order to prove such a relation it is necessary to show that sufficient injury has caused a lesion of the dura or pia with affection of the brain tissue Evidences of old hemorrhage with adhesion of brain to membranes beneath the point of injury are the only satisfactory data that can be offered as proof Such cases have been fully reported by Hitzig, Eppinger Thomas and Bartlett, Keen, Annadale Adler, Cassassa and others. Both sarcoma and glioma are observed under such conditions Somewhat less satisfactory and more numerous are the cases in which the tumor does not appear at the point of injury but owing to the influence of contrecoup

the brain lesion may be far removed from the point of the blow. Additional evidence may appear in the clinical history when it is shown that the tumor symptoms are continuous with, or appeared shortly after the injury. With increasing intervals between trauma and tumor, the relation must be regarded as less direct. Starr's assumption that a cerebral hemorrhage or contusion may pass directly into a tumor process is rejected by Oppenheim and Bruns, but such an interpretation is fully in accord with histological findings as well as clinical history. In the organization of a cerebral blood clot, the growth of capillaries and glia tissue may be active."

Cushing's opinion was as follows: "The relation between trauma and the first appearance of symptoms is a coincidence that occurs too often to be ignored. And not infrequently, on shaving a patient's scalp, the scar of an old and forgotten injury may even indicate the precise situation of the lesion, this being particularly true of meningeal tumors. It must, however, be admitted that a latent tumor itself, through an epileptic attack or faint, may have been the cause of the fall producing the cranial injury. Hence, trauma may not actually occasion, but may only serve to bring a pre-existing lesion into symptomatic prominence, owing possibly to the rupture of a blood vessel in a vascular growth."
S. A. K. Wilson denies any relationship between trauma and tumor of the central nervous system, basing his opinion on his experience with head injuries sustained in the World War.

Percival Bailey in his recent book, "Intracranial Tumors," expresses the opinion that "it is much more probable that the trauma serves merely to call attention to a pre-existing tumor by aggravating its symptoms."

In a recent contribution on the subject by Weinberg in which the literature is thoroughly reviewed and a series of

102 brain tumor cases analyzed, the opinion is offered that a relationship does exist between trauma and brain tumor either causation or activation of a latent brain neoplasm In his series of cases, Weinberg found that 7.8 per cent fulfilled the strict requirements necessary to establish a relationship between trauma and tumor He discusses the controversy on this problem which reached an active stage in Germany in 1932. At that time Beneke maintained that a direct relationship existed between head injury and glioma of the brain His theory was based on the tendency of reactive glial cells which increase in necrotic areas of the brain to become malignant The necrotic areas are due to ischemic changes in the brain as a result of injury, the mechanism of the ischemia being a vascular one, either anatomical in nature (actual vascular injury) or physiological (arterial spasm) These reactive glial elements he feels go on to irritate adjacent cells and result in tumor forms.

Borst agrees with Beneke and adds that due to trauma embryonal cell layers may be affected and the neoplasm precipitated Borst in reporting the pathological observations in the brain of a tumor case tells of finding tumor tissue surrounding an area of softening. Because in certain areas he found brain tissue and not tumor tissue bordering on the area of softening he felt justified in assuming that he was dealing with a condition in which the tumor was secondary to the softening

Reports in the literature of similar studies to establish pathological evidence either for or against the etiological relationship of trauma to brain tumor are exceedingly rare. In a careful search of our own records we have been able to find several cases in which there seemed to be a rather direct relationship between the injury and the development of a neoplasm One patient developed a large meningeal

tumor directly under the scar of a scalp laceration which marked the site of a previous head trauma (Figs 93 and 94) Another patient began having Jacksonian motor convulsions



Fig 93 Large meningioma with cranial hyperostosis which developed at the site of a previous head trauma



Fig 94 Wound ten days following removal of tumor Note scar of preceding trauma in center of scalp flap

eight weeks after a head trauma. These persisted until a craniotomy was done several months later At operation a large glioma was found directly under the scar in the scalp which indicated the site of the previous injury

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